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Welcome to STN International! Enter x:
X
Welcome to STN International! Enter x:
LOGINID: ssptacmb1647
PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2
                      Welcome to STN International
                  Web Page URLs for STN Seminar Schedule - N. America
 NEWS
                  "Ask CAS" for self-help around the clock
 NEWS
                  The Derwent World Patents Index suite of databases on STN
          OCT 23
 NEWS 3
                  has been enhanced and reloaded
          OCT 30 CHEMLIST enhanced with new search and display field
 NEWS 4
                  JAPIO enhanced with IPC 8 features and functionality
          NOV 03
 NEWS
          NOV 10 CA/CAplus F-Term thesaurus enhanced
 NEWS
                  STN Express with Discover! free maintenance release Version
          NOV 10
 NEWS 7
                  8.01c now available
                  CAS Registry Number crossover limit increased to 300,000 in
          NOV 20
 NEWS
                  additional databases
                  CA/CAplus to MARPAT accession number crossover limit increased
          NOV 20
 NEWS
                  to 50,000
 NEWS 10 DEC 01 CAS REGISTRY updated with new ambiguity codes
                  CAS REGISTRY chemical nomenclature enhanced
          DEC 11
 NEWS 11
                  WPIDS/WPINDEX/WPIX manual codes updated
          DEC 14
 NEWS 12
          DEC 14 GBFULL and FRFULL enhanced with IPC 8 features and
 NEWS 13
                  functionality
          DEC 18 CA/CAplus pre-1967 chemical substance index entries enhanced
 NEWS 14
                  with preparation role
                  CA/CAplus patent kind codes updated
 NEWS 15
          DEC 18
                  MARPAT to CA/CAplus accession number crossover limit increased
 NEWS 16 DEC 18
                  to 50,000
                  MEDLINE updated in preparation for 2007 reload
          DEC 18
 NEWS 17
          DEC 27 CA/CAplus enhanced with more pre-1907 records
 NEWS 18
          JAN 08 CHEMLIST enhanced with New Zealand Inventory of Chemicals
 NEWS 19
 NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.
               STN Operating Hours Plus Help Desk Availability
 NEWS HOURS
               Welcome Banner and News Items
 NEWS LOGIN
               For general information regarding STN implementation of IPC 8
 NEWS IPC8
               X.25 communication option no longer available
 NEWS X25
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Enter NEWS followed by the item number or name to see news on that specific topic.

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FILE 'HOME' ENTERED AT 11:10:17 ON 11 JAN 2007
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TITLE:

=> file medline embase biosis caplus COST IN U.S. DOLLARS SINCE FILE TOTAL ENTRY SESSION 0.42 0.42 FULL ESTIMATED COST FILE 'MEDLINE' ENTERED AT 11:11:10 ON 11 JAN 2007 FILE 'EMBASE' ENTERED AT 11:11:10 ON 11 JAN 2007 Copyright (c) 2007 Elsevier B.V. All rights reserved. FILE 'BIOSIS' ENTERED AT 11:11:10 ON 11 JAN 2007 Copyright (c) 2007 The Thomson Corporation FILE 'CAPLUS' ENTERED AT 11:11:10 ON 11 JAN 2007 USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT. PLEASE SEE "HELP USAGETERMS" FOR DETAILS. COPYRIGHT (C) 2007 AMERICAN CHEMICAL SOCIETY (ACS) => s granulocyte(w) macrophage(w) colony(w) stimulating(w) factor or granulocyte(w)colony(w)stimulating(w)factor 112622 GRANULOCYTE(W) MACROPHAGE(W) COLONY(W) STIMULATING(W) FACTOR OR GRANULOCYTE (W) COLONY (W) STIMULATING (W) FACTOR => s ll and (ischemia or hypoxia or Parkinson? or stroke or amyotrophic(w)latéral(w)sclerosis or ALS or Lou(w)Gehrig) 1307 L1 AND (ISCHEMIA OR HYPOXIA OR PARKINSON? OR STROKE OR AMYOTROPH L2IC(W) LATERAL(W) SCLEROSIS OR ALS OR LOU(W) GEHRIG) => s 12 and erythropoietin or interleuikin 134 L2 AND ERYTHROPOIETIN OR INTERLEUIKIN L3=> s 13 and (tpa or tissue(w)plasminogen(w)activator) 4 L3 AND (TPA OR TISSUE(W) PLASMINOGEN(W) ACTIVATOR) L4=> dup rem 14 PROCESSING COMPLETED FOR L4 4 DUP REM L4 (0 DUPLICATES REMOVED) L5 => dup rem 13 PROCESSING COMPLETED FOR L3 113 DUP REM L3 (21 DUPLICATES REMOVED) => dis his (FILE 'HOME' ENTERED AT 11:10:17 ON 11 JAN 2007) FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 11:11:10 ON 11 JAN 2007 112622 S GRANULOCYTE (W) MACROPHAGE (W) COLONY (W) STIMULATING (W) FACTOR OR G L11307 S L1 AND (ISCHEMIA OR HYPOXIA OR PARKINSON? OR STROKE OR AMYOTR L2134 S L2 AND ERYTHROPOIETIN OR INTERLEUIKIN L34 S L3 AND (TPA OR TISSUE(W)PLASMINOGEN(W)ACTIVATOR) L44 DUP REM L4 (0 DUPLICATES REMOVED) L5 113 DUP REM L3 (21 DUPLICATES REMOVED) L6 => dis ibib abs 15 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN L5 ACCESSION NUMBER: 2005:1265316 CAPLUS DOCUMENT NUMBER: 144:17858

expression in mammals

Expression vector with regulatory elements for gene

INVENTOR(S):

Webster, Keith A.

PATENT ASSIGNEE(S):

USA

SOURCE:

U.S. Pat. Appl. Publ., 24 pp., Cont.-in-part of U.S.

Ser. No. 723,326.

CODEN: USXXCO

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PATENT NO. KIND DATE APPLICATION NO. DATE ----20051201 US 2005-118963 A1 20050429 US 2005266549 20050517 US 2000-723326 20001128 US 1999-171597P P 19991223 US 6893867 B1 PRIORITY APPLN. INFO.: US 2000-723326 A2 20001128

Expression vectors are disclosed that are comprised of (a) one or more AB silencer elements and conditionally inducible elements to form silencer-inducible regions and (b) promoters in operative linkage upstream of at least one expressed region. The expression vector thereby regulates expression of at least one downstream region by conditional silencing in which an expressed DNA region of a gene is transcribed to produce RNA transcripts, which may or may not be translated to produce polypeptides. Genetically engineered mammalian cells and non-human mammals can be made using such expression vectors through transfection and transgenic techniques. Moreover, processes of making and using the aforementioned products are disclosed (e.g., the expression vector may be used diagnostically, therapeutically, or prophylactically).

=> dis ibib abs 15 2-4

ANSWER 2 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN L5

ACCESSION NUMBER:

2005:572333 CAPLUS

DOCUMENT NUMBER:

143:91472

TITLE:

Methods of treating neurological conditions with

hematopoietic growth factors

INVENTOR(S):

Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger, Carola; Sommer, Clemens; Schwab, Stefan; Kollmar, Rainer; Maurer, Martin; Weber, Daniela; Gassler,

Nikolaus

PATENT ASSIGNEE(S):

Axaron Bioscience Ag, Germany

SOURCE:

U.S. Pat. Appl. Publ., 169 pp., Cont.-in-part of Appl.

No. PCT/IB03/06446.

CODEN: USXXCO

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PATENT	PATENT NO. KIND) 1	DATE		ž	APPL	ICAT:	ION 1	NO.		DA	ATE	
					_			,						~ -		
US 2005	1421	02		Al		2005	0630	1	US 20	004-	8801	01		20	040	530
US 2004	1419	46	-	A1		2004	0722	1	US 20	003-	6592	95		20	0030	911
WO 2004	0582	87		A2		2004	0715	1	WO 2	003-	IB64	4 6 [.]		20	0031	231
WO 2004	WO 2004058287 A8					2004	1021									
WO 2004058287 A3				2004	1216			ŕ								
W:	ΑE,	AG,	AL,	AM,	AT,	AU,	AZ,	BA,	BB,	BG,	BR,	BW,	BY,	BZ,	CA,	CH,
	CN,	CO,	CR,	CU,	CZ,	DE,	DK,	DM,	DZ,	EC,	EE,	EG,	ES,	FΙ,	GB,	GD,
	GE,	GH,	GM,	HR,	HU,	ID,	IL,	IN,	IS,	JP,	KE,	KG,	KP,	KR,	KZ,	LC,
	LK,	LR,	LS,	LT,	LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NI,	NO,
	NZ,	OM,	PG,	PH,	PL,	PT,	RO,	RU,	SC,	SD,	SE,	SG,	SK,	SL,	SY,	TJ,
	TM,	TN,	TR,	TT,	TZ,	UA,	UG,	US,	UΖ,	VC,	VN,	YU,	ZA,	ZM,	ZW	
RW:	BW,	GH,	GM,	KE,	LS,	MW,	MZ,	SD,	SL,	SZ,	TZ,	UG,	ZM,	ZW,	AM,	AZ,

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BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
             TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     WO 2006008582
                                20060126
                                            WO 2004-IB4329
                                                                   20041229
                          A1
            AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM,
             SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
        RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF,
             CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM,
             KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG,
             KZ, MD, RU, TJ, TM
                                            US 2002-331755
                                                                B1 20021231
PRIORITY APPLN. INFO.:
                                                                A2 20030911
                                            US 2003-659295
                                                                A2 20031231
                                            WO 2003-IB6446
                                            US 2004-880101
                                                                A 20040630
     The present invention relates to a method of treating a neurol. condition
AB
     in a mammal by administering at least one hematopoietic growth factor from
     the group consisting of GCSF, GMCSF, IL-3, IL-5, a derivative thereof, or a
     mimetic thereof. A method is also claimed of treating a neurol. condition
     using neural stem cells treated with a hematopoietic factor. Also claimed
     is a method of enhancing the survival of a cell transplanted into a
     mammal, comprising introducing into the cell one or more polynucleotides
     which encode a hematopoietic factor. A method of enhancing the viability
     of a neural cell culture comprising contacting the neural cell culture
     with a hematopoietic factor is addnl. claimed.
                    CAPLUS COPYRIGHT 2007 ACS on STN
     ANSWER 3 OF 4
                         2004:565109 CAPLUS
                         141:100449
```

L5

ACCESSION NUMBER:

DOCUMENT NUMBER:

TITLE:

Methods of treating neurological conditions with

hematopoietic growth factors

INVENTOR(S):

Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger, Carola; Sommer, Clemens; Schwab, Stefan; Kollmar, Rainer; Maurer, Martin; Weber, Daniela; Gassler,

Nikolaus

PATENT ASSIGNEE(S):

Axaron Bioscience AG, Germany

SOURCE:

PCT Int. Appl., 210 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE				
WO 2004058287	A2	20040715	WO 2003-IB6446	20031231				
WO 2004058287	A8 .	20040713	WO 2003 1D0110	2003200				
WO 2004058287	A3	20041216						
W: AE, AG,	AL, AM, A	Γ, AU, AZ,	BA, BB, BG, BR, BW,	BY, BZ, CA, CH,				
CN, CO,	CR; CU, CZ	Z, DE, DK,	DM, DZ, EC, EE, EG,	ES, FI, GB, GD,				
			IN, IS, JP, KE, KG,	_				
LK, LR	LS, LT, L	U, LV, MA,	MD, MG, MK, MN, MW,	MX, MZ, NI, NO,				
· ·			RU, SC, SD, SE, SG,					
TM, TN,	TR, TT, T	Z, UA, UG,	US, UZ, VC, VN, YU,	ZA, ZM, ZW				
RW: BW, GH,	GM, KE, LS	S, MW, MZ,	SD, SL, SZ, TZ, UG,	ZM, ZW, AM, AZ,				
			AT, BE, BG, CH, CY,					
ES, FI,	FR, GB, GI	R, HU, IE,	IT, LU, MC, NL, PT,	RO, SE, SI, SK,				
TR, BF	BJ, CF, CO	G, CI, CM,	GA, GN, GQ, GW, ML,	MR, NE, SN, TD, TG				
US 2004141946	A1							
CA 2511294	Al	20040715	40715 CA 2003-2511294 20031231					

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AU 2003-299430
    AU 2003299430
                                20040722
                                                                  20031231
                         A1
                                20051005
                                           EP 2003-799727
                         A2
                                                                  20031231
    EP 1581249
        R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
                                           BR 2003-17910
    BR 2003017910
                         A
                               20051129
                                                                  20031231
                                           CN 2003-80110075
                               20060405
    CN 1756556
                         A
                                                                  20031231
                                           JP 2005-509731
    JP 2006512419
                         T
                               20060413
                                                                  20031231
    US 2005142102
                         Al
                               20050630
                                           US 2004-880101
                                                                  20040630
PRIORITY APPLN. INFO.:
                                           US 2002-331755
                                                               A 20021231
                                           US 2003-659295
                                                               A 20030911
                                           WO 2003-IB6446
                                                               W 20031231
```

The present invention relates to a method of treating neurol. conditions AB in a mammal by administering a hematopoietic growth factor such as granulocyte-colony stimulating factor (GCSF) and granulocyte-macrophage colony stimulating factor (GMCSF). The invention also provides methods of screening for compds. that bind to a GCSF or GMCSF receptor found on the surface of a neuronal cell; and which provides a neuroprotective, neuroproliferative and/or a STAT gene activation activity.

ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN L5

2003:282718 CAPLUS ACCESSION NUMBER:

DOCUMENT NUMBER: 138:282352

Traversal of nucleic acid molecules through a tissue TITLE:

fluid space and expression in repair cells

Sosnowski, Barbara A.; Pierce, Glenn INVENTOR(S):

Selective Genetics, Inc., USA PATENT ASSIGNEE(S): PCT Int. Appl., 95 pp.

SOURCE: CODEN: PIXXD2

DOCUMENT TYPE: Patent English LANGUAGE:

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PA.	PATENT NO.				KINI		DATE		i	APPL:	ICAT:	ION I	NO.		D	ATE	
WO	2003	0294	29				2003	0410	Ţ	WO 2	002-1	US31	546		20	0021	002
WO	2003	0294	29		A3		2004	0401									
WO	2003	0294	29		A9		2004	0701									
	W:	AE,	AG,	AL,	AM,	AT,	AU,	AZ,	BA,	BB,	BG,	BR,	BY,	BZ,	CA,	CH,	CN,
		CO,	CR,	CU,	CZ,	DE,	DK,	DM,	DZ,	EC,	EE,	ES,	FI,	GB,	GD,	GE,	GH,
		GM,	HR,	HU,	ID,	IL	IN,	IS,	JP,	KE,	KG,	KP,	KR,	KZ,	LC,	LK,	LR,
		LS,	LT,	LU,	LV,	MA	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NO,	ΝŹ,	OM,	PH,
		PL,	PT,	RO,	RU,	SD	SE,	SG,	SI,	SK,	SL,	TJ,	TM,	TN,	TR,	TT,	TZ,
		UA,	UG,	US,	UZ,	VC,	VN,	YU,	ZA,	ZM,	ZW						
•	RW:	GH;	GM,	KE,	LS,	MW.	MZ,	SD,	SL,	SZ,	TZ,	UG,	ZM,	ZW,	AM,	AZ,	BY,
		KG,	KZ,	MD,	RU,	TJ,	TM,	AT,	BE,	BG,	CH,	CY,	CZ,	DE,	DK,	EE,	ES,
		FI,	FR,	GB,	GR,	IE,	IT,	LU,	MC,	NL,	PT,	SE,	SK,	TR,	BF,	ВJ,	CF,
		CG,	CI,	CM,	GA,	GN,	GQ,	GW,	ML,	MR,	NE,	SN,	TD,	TG			
AU	2002	3434	75		A1		2003	0414		AU 2	002-	3434	75		2	0021	002
US	2003	1489	79		A1		2003	0807	1	US 2	002-	2642	84		2	0021	002
EP	1438	413			A2		2004	0721	;	EP 2	002-	7804	19		2	0021	002
	R:	AT,	BE,	CH,	DE,	DK,	ES,	FR,	GB,	GR,	IT,	LI,	LU,	NL,	SE,	MC,	PT,
		IE,	SI,	LT,	LV,	FI	RO,	MK,	CY,	AL,	TR,	BG,	CZ,	ĒE,	SK		
PRIORITY	Y APP				٠,								13P			0011	003
									1	WO 2	002-	US31	546	1	W 2	0021	002

Disclosed are methods for use in transferring nucleic acids into cells at AB a wound site associated with a fluid space. These gene transfer protocols are suitable for use in transferring various nucleic acids into cartilage, cardiac muscle, and other tissues, and have many uses including treating diseases such as arthritis and ischemic heart disease, and promoting wound healing. The invention further disclosed pharmaceutical compns. that may be used in the practice of the invention to transfer the nucleic acid of

interest. Such compns. include any multi-partitioned biocompatible matrix in combination with multiple nucleic acids of interest. Thus, collagen collagen-immobilized fibroblast growth factor (FGF) genes induce angiogenesis in vitro, and FGF gene delivery to skeletal muscle wounds induces both angiogenesis and arteriogenesis and well as induces myocyte regeneration.

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FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 11:11:10 ON 11 JAN 2007

112622 S GRANULOCYTE(W) MACROPHAGE(W) COLONY(W) STIMULATING(W) FACTOR OR G

1307 S L1 AND (ISCHEMIA OR HYPOXIA OR PARKINSON? OR STROKE OR AMYOTR

134 S L2 AND ERYTHROPOIETIN OR INTERLEUIKIN

4 S L3 AND (TPA OR TISSUE(W) PLASMINOGEN(W) ACTIVATOR)

4 DUP REM L4 (0 DUPLICATES REMOVED)

113 DUP REM L3 (21 DUPLICATES REMOVED)

=> dis ibib abs 16 100-113

L6 ANSWER 100 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 96235644 EMBASE

DOCUMENT NUMBER:

1996235644

TITLE:

Possible role of tumor necrosis factor-alpha in erythropoietic suppression by endotoxin and

granulocyte/macrophage colony-

stimulating factor.

AUTHOR:

Udupa K.B.; Sharma B.G.

CORPORATE SOURCE:

Dr. K.B. Udupa, VA Medical Center, 4300 West Seventh

Street, Little Rock, AR 72205, United States

SOURCE:

American Journal of Hematology, (1996) Vol. 52, No. 3, pp.

178-183. .

ISSN: 0361-8609 CODEN: AJHEDD

COUNTRY:
DOCUMENT TYPE:

FILE SEGMENT:

United States

Journal; Article

025 Hematology

026 Immunology, Serology and Transplantation

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE:

L6

STN

Entered STN: 18 Jun 1997

Last Updated on STN: 18 Jun 1997

AB Injection of bacterial endotoxin or granulocyte/

macrophage colony-stimulating factor

(GM-CSF) into exhypoxic polycythemic mice simultaneously with erythropoietin (EPO) suppressed erythroid cell formation, as monitored by 59Fe incorporation into circulating red blood cells. This effect was dose-dependent and time-dependent, GM CSF did not inhibit erythroid cell formation directly, as the antibody to the GM-CSF did not neutralize the effect of endotoxin, the inducer of GM-CSF. The suppression of both agents could be partially corrected by prior injection of a monoclonal antibody to tumor necrosis factor α (anti-TNF α). These results indicate that the suppression of EPO-induced erythroid cell formation by endotoxin and GM-CSF was due in part to the production of TNF α .

ANSWER 101 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

ACCESSION NUMBER: 1996:440012 BIOSIS DOCUMENT NUMBER: PREV199699162368

TITLE: Biology and pathophysiology of leukotrienes.

AUTHOR(S): Denzlinger, Claudio

CORPORATE SOURCE: Med. Klinik III, Klinikum Grosshadern, Ludwig-Maximilians

Univ. Muenchen, 81377 Muenchen, Germany

SOURCE: Critical Reviews in Oncology-Hematology, (1996) Vol. 23,

No. 3, pp. 167-223.

ISSN: 1040-8428.

DOCUMENT TYPE: Article

General Review; (Literature Review)

LANGUAGE: English

ENTRY DATE: Entered STN: 7 Oct 1996

Last Updated on STN: 7 Oct 1996

L6 ANSWER 102 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights

reserved on STN

ACCESSION NUMBER: 96158763 EMBASE

DOCUMENT NUMBER:

1996158763

TITLE:

Potential role of hemopoietic cytokines in neuronal

survival.

AUTHOR: Sigel K.; Rosenbaum D.M.

CORPORATE SOURCE: Dept. of Neurology, Albert Einstein College of Medicine,

1300 Morris Park Ave., Bronx, NY 10461, United States

SOURCE: Drug News and Perspectives, (1996) Vol. 9, No. 3, pp.

142-148. .

ISSN: 0214-0934 CODEN: DNPEED

COUNTRY:

Spain

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT:

002 Physiology

005 General Pathology and Pathological Anatomy

١

008 Neurology and Neurosurgery

025 Hematology

029 Clinical Biochemistry

030 Pharmacology

037 Drug Literature Index

LANGUAGE:

English

ENTRY DATE: Entered STN: 3 Jul 1996

Last Updated on STN: 3 Jul 1996

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 103 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights

reserved on STN

ACCESSION NUMBER: . 95334463 EMBASE

DOCUMENT NUMBER:

1995334463

TITLE: Treatment with retinoids and haemopoietic growth factors in

myelodysplastic syndromes [1].

AUTHOR: Ciotti R.; Rosti V.; Lucotti C.; Forloni F.; Romeo G.;

Pezzoli A.

CORPORATE SOURCE: First Div. of Internal Medicine, Ospedale Consorziale,

Azienda Sanitaria 13,24047 Treviglio, Italy

SOURCE: British Journal of Haematology, (1995) Vol. 91, No. 3, pp.

773-774. .

ISSN: 0007-1048 CODEN: BJHEAL

COUNTRY: United Kingdom DOCUMENT TYPE: Journal; Letter

Journal; Letter 016 Cancer

025 Hematology

037 Drug Literature Index038 Adverse Reactions Titles

LANGUAGE:

FILE SEGMENT:

English

ENTRY DATE: Entered STN: 5 Dec 1995

Last Updated on STN: 5 Dec 1995

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 104 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN

ACCESSION NUMBER: 1995:505151 BIOSIS

DOCUMENT NUMBER: PREV199598510201

TITLE: Erythropoietin and deferred autologous blood

collection: From physiological secretion to the rationale

for exogenous supplementation.

AUTHOR(S): Casadevall, N.

CORPORATE SOURCE: Hop. Raymond Poincare, Lab. Hematol., 104 blvd. Raymond

Poincare, F-92380 Garches, France

SOURCE: Nouvelle Revue Française d'Hematologie, (1995) Vol. 37, No.

SUPPL. 1, pp. S11-S15.

CODEN: NRFHA4. ISSN: 0029-4810.

DOCUMENT TYPE: Article

General Review; (Literature Review)

LANGUAGE: French

ENTRY DATE: Entered STN: 29 Nov 1995

Last Updated on STN: 29 Nov 1995

L6 ANSWER 105 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1994:699076 CAPLUS

DOCUMENT NUMBER: 121:299076

TITLE: The kinetoplastid membrane protein 11 of Leishmania

donovani and African trypanosomes is a potent

stimulator of T-lymphocyte proliferation

AUTHOR(S): Tolson, Douglas L.; Jardim, Armando; Schnur, Lionel

F.; Stebeck, Caroline; Tuckey, Corinna; Beecroft,

Robert P.; Teh, Hung-Sia; Olafson, Robert W.; Pearson,

Terry W.

CORPORATE SOURCE: Dep. Biochem. Microbiol., Univ. Victoria, Victoria,

BC, V8W 3P6, Can.

SOURCE: Infection and Immunity (1994), 62(11), 4893-9

CODEN: INFIBR; ISSN: 0019-9567

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal LANGUAGE: English

Kinetoplastid membrane protein 11 (KMP-11) from Leishmania donovani is an AB abundant 11-kDa surface membrane glycoprotein. Lymph node cells from mice of six different H-2 haplotypes immunized with KMP-11 or with L. donovani promastigotes were stimulated to proliferate in vitro with purified KMP-11. Primed purified T cells required antigen presentation since they were not stimulated unless KMP-11-pulsed or L. donovani-infected macrophages were added. Promastigotes of a wide variety of Leishmania species and procyclic forms of African trypanosomes stimulated proliferation of KMP-11-primed or L. donovani promastigote-primed lymph node cells. All of the Leishmania promastigotes and African trypanosomes tested contained an 11-kDa protein, as detected by immunoblotting with KMP-11-specific monoclonal antibodies. The widespread distribution of the 11-kDa (KMP-11) mols. and their ability to stimulate strong T-lymphocyte proliferation in a non-H-restricted fashion suggest that they may be important mols. for induction of cell-mediated immune responses.

L6 ANSWER 106 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 94115823 EMBASE

DOCUMENT NUMBER: 1994115823

SOURCE:

TITLE: Evidence suggesting a negative regulatory role for

macrophages in murine erythropoiesis in vivo.

AUTHOR: Wang C.Q.; Udupa K.B.; Xiao H.; Lipschitz D.A.

CORPORATE SOURCE: John L. McClelland, Memorial Veterans Hospital, 4300 West

Seventh Street, Little Rock, AR 72205, United States Experimental Hematology, (1994) Vol. 22, No. 4, pp.

370-376. .

ISSN: 0301-472X CODEN: EXHEBH

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 025 Hematology

037 Drug Literature Index

LANGUAGE: English
SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 25 May 1994

Last Updated on STN: 25 May 1994

Increasing the rate of erythropoiesis in C57BL/6 mice, either by AB hypoxia or by the injection of recombinant erythropoietin (Epo), resulted in significant reductions in marrow macrophage number, as assessed by flow cytometry employing the monoclonal antibody against the macrophage antigen Mac-1 and by histologic determination of reductions in the number of marrow esterase-positive cells. This decline was paralleled by decreases in marrow colony-forming unit-macrophage (CFU-M) and colony-forming unit-granulocyte/macrophage (CFU-GM) number. The intramedullary concentration of the cytokines interleukin- 1α (IL-la) and tumor necrosis factor-a (TNF-a), which are produced by macrophages, was also reduced. Cessation of erythropoiesis was associated with increases in macrophage number, CFU-M and CFU-GM colony number, and $IL-1\alpha$ concentrations. Increased erythropoiesis resulted in reductions in number of burst-forming unit-erythroid (BFU-E) colonies, which were less sensitive to suppression by macrophages as evidenced by less increase in colony number when macrophages were removed from the marrow before in vitro BFU-E culture. BFU-E colony number was suppressed less when IL-1 α and TNF- α were added to cultures obtained from animals with stimulated erythropoiesis. Compared to controls, BFU-E number and suppression by macrophages increased significantly when erythropoiesis was reduced. These observations provide compelling evidence for a regulatory role for macrophages in normal erythropoiesis in vivo, presumably acting as a negative balance to the stimulatory effects of Epo.

L6 ANSWER 107 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 94010380 EMBASE

DOCUMENT NUMBER: 1994010380

TITLE: Recombinant human DNase for treatment of cystic fibrosis.

AUTHOR: Wordell C.J.

CORPORATE SOURCE: Drug Information Service, Department of Pharmacy, Thomas

Jefferson University Hospital, 11th and Walnut Streets, Philadelphia, PA 19107, United States Hospital Pharmacy, (1993) Vol. 28, No. 12, pp.

1226+1229-1232+1240. .

ISSN: 0018-5787 CODEN: HOPHAZ

COUNTRY: United States
DOCUMENT TYPE: Journal; Note

FILE SEGMENT: 006 Internal Medicine

Ol5 Chest Diseases, Thoracic Surgery and Tuberculosis

037 Drug Literature Index

LANGUAGE: English

SOURCE:

ENTRY DATE: Entered STN: 30 Jan 1994

Last Updated on STN: 30 Jan 1994

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 108 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 93080744 EMBASE

DOCUMENT NUMBER: 1993080744

TITLE: [Hematopoietic growth hormone factors as adjuncts in

antiretroviral therapy].

HAMATOPOETISCHE WACHSTUMSFAKTOREN ALS

ZUSATZBEHANDLUNG BEI DER ANTIRETROVIRALEN THERAPIE. AIDS-Forschung, (1993) Vol. 8, No. 2, pp. 69-70.

SOURCE: AIDS-Forschung, (1993) Vol. 8, ISSN: 0179-3098 CODEN: AIFOER

COUNTRY: Germany

DOCUMENT TYPE: Journal; Note

FILE SEGMENT: 004 Microbiology

025 Hematology

026 Immunology, Serology and Transplantation

037 Drug Literature Index038 Adverse Reactions Titles

LANGUAGE: German

ENTRY DATE: Entered STN: 18 Apr 1993

Last Updated on STN: 18 Apr 1993

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 109 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights

reserved on STN

ACCESSION NUMBER: 93073784 EMBASE

DOCUMENT NUMBER: 1993073784

TITLE: Stimulating new developments: Colony-stimulating factors.

AUTHOR: Kare D.

CORPORATE SOURCE: Home Nutritional Services, Chicago, IL, United States

SOURCE: Journal of Intravenous Nursing, (1993) Vol. 16, No. 1, pp.

37-43. .

ISSN: 0896-5846 CODEN: JINUEE

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 025 Hematology

037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 11 Apr 1993

Last Updated on STN: 11 Apr 1993

Drug-induced low white-blood-cell counts have long impaired our ability to treat patients. Colony-stimulating factors are now available for intravenous or subcutaneous administration. These glycoproteins act on hematopoietic cells by binding to specific cell surface receptors and by stimulating proliferation, differentiation, commitment, and activation of new white blood cells. A brief overview of patient population, indications, actions, and adverse reactions for hospital or home use is

indications, actions, and adverse reactions for hospital or home use is presented.

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reserved on STN

ACCESSION NUMBER: 92284673 EMBASE DOCUMENT NUMBER: 1992284673

ANSWER 110 OF 113

L6

DOCUMENT NUMBER: 1992284673
TITLE: [Growth factors in hematology].

GROEIFACTOREN IN DE HEMATOLOGIE.

AUTHOR: Demyunck H.; Boogaerts M.A.

CORPORATE SOURCE: Afdeling Hematologie, Universitaire Ziekenhuizen,

Katholieke Universiteit, Leuven, Belgium

SOURCE: Tijdschrift voor Geneeskunde, (1992) Vol. 48, No. 16, pp.

1187-1196. .

ISSN: 0371-683X CODEN: TGEKBW

COUNTRY: Belgium

DOCUMENT TYPE: Journal; (Short Survey)

FILE SEGMENT: 016 Cancer 025 Hematology

026 Immunology, Serology and Transplantation

029 Clinical Biochemistry

030 Pharmacology

037 Drug Literature Index 038 Adverse Reactions Titles

LANGUAGE: Dutch

ENTRY DATE: Entered STN: 25 Oct 1992

Last Updated on STN: 25 Oct 1992

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

ANSWER 111 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6

reserved on STN

92343779 EMBASE ACCESSION NUMBER:

DOCUMENT NUMBER:

1992343779

Regulation of erythropoiesis in the newborn: A complex TITLE:

system.

Heikinheimo M.; Siimes M.A. **AUTHOR:**

The Children's Hospital, University of Helsinki, SF-00290 CORPORATE SOURCE:

Helsinki, Finland

Annals of Medicine, (1992) Vol. 24, No. 5, pp. 309-311. . SOURCE:

ISSN: 0785-3890 CODEN: ANMDEU

United Kingdom COUNTRY: Journal; Editorial DOCUMENT TYPE:

Pediatrics and Pediatric Surgery 007 FILE SEGMENT:

> Obstetrics and Gynecology 010

Developmental Biology and Teratology 021

Hematology 025

Clinical Biochemistry 029 Drug Literature Index 037

English LANGUAGE:

Entered STN: 13 Dec 1992 ENTRY DATE:

Last Updated on STN: 13 Dec 1992

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

DUPLICATE 10 MEDLINE on STN L6 ANSWER 112 OF 113

91358449 MEDLINE ACCESSION NUMBER: PubMed ID: 1653242 DOCUMENT NUMBER:

TITLE:

Hypoxia up-regulates the activity of a novel erythropoietin mRNA binding protein.

Rondon I J; MacMillan L A; Beckman B S; Goldberg M A; **AUTHOR:**

Schneider T; Bunn H F; Malter J S

Department of Pharmacology, Tulane University School of CORPORATE SOURCE:

Medicine, New Orleans, Louisiana 70112.

CA-01427 (NCI) CONTRACT NUMBER:

DK-01401 (NIDDK) DK-41234 (NIDDK)

The Journal of biological chemistry, (1991 Sep 5) Vol. 266, SOURCE:

No. 25, pp. 16594-8.

Journal code: 2985121R. ISSN: 0021-9258.

United States PUB. COUNTRY:

Journal; Article; (JOURNAL ARTICLE) DOCUMENT TYPE:

English LANGUAGE:

Priority Journals FILE SEGMENT:

199110 ENTRY MONTH:

Entered STN: 27 Oct 1991 ENTRY DATE:

> Last Updated on STN: 3 Feb 1997 Entered Medline: 4 Oct 1991

AB The mechanisms which control the production of erythropoietin (Epo) remain enigmatic. Recent data suggest that the half-time of Epo messenger RNA (mRNA) is increased by hypoxia in Hep 3B cells, a human hepatoma line. The post-transcriptional regulation of other rapidly degraded mRNAs is mediated by sequence-specific mRNA binding proteins. In order to determine if Epo mRNA specific binding proteins exist, we probed cytosolic lysates from Hep 3B cells and mouse tissues with radiolabeled Epo RNA. A cytosolic protein that binds specifically to Epo RNA was identified in the Epo-producing, hepatoblastoma Hep 3B cell line by gel mobility shift assay. This protein was identified in both normoxic and hypoxic cells and bound specifically to a 120-base fragment of the 3'-untranslated region (3'-UTR) of Epo mRNA. Binding was completed with . unlabeled Epo RNA, but not with granulocyte-macrophage colony-stimulating factor RNA. Ultraviolet light cross-linked Epo RNA-protein complexes migrated as two bands of 70

and 135-140 kD on sodium dodecyl sulfate-polyacrylamide gels. Binding activity was markedly increased in brain and spleen lysates from mice

subjected to 24 h of hypoxia. Therefore, the post-transcriptional regulation of Epo expression in response to hypoxia may in part be due to the interaction of Epo RNA with its specific binding protein.

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reserved on STN

88272110 EMBASE ACCESSION NUMBER:

DOCUMENT NUMBER:

1988272110

TITLE:

Up-regulation of interleukin 4/B-cell stimulatory factor 1

receptor expression.

AUTHOR:

Ohara J.; Paul W.E.

CORPORATE SOURCE:

Laboratory of Immunology, National Institute of Allergy and

Infectious Diseases, National Institutes of Health,

Bethesda, MD 20892, United States

SOURCE:

Proceedings of the National Academy of Sciences of the United States of America, (1988) Vol. 85, No. 21, pp.

8221-8225. .

ISSN: 0027-8424 CODEN: PNASA6

COUNTRY:

United States

DOCUMENT TYPE:

Journal

FILE SEGMENT:

Immunology, Serology and Transplantation 026

LANGUAGE: SUMMARY LANGUAGE: English

English

ENTRY DATE:

Entered STN: 11 Dec 1991

Last Updated on STN: 11 Dec 1991

The expression of interleuikin 4 (IL-4) receptors on resting T AB and B lymphocytes was enhanced 4- to 8-fold by IL-4 stimulation of these cells. Other agents such as lipopolysaccharide and anti-IgM for B cells and concanavalin A for T cells also caused increased IL-4 receptor expression, although to a somewhat smaller degree than IL-4. Using a newly developed flow cytometric analysis based on the binding of biotinylated IL-4 and phycoerythrin-streptavidin, it was observed that receptor up-regulation in a T-cell population treated with IL-4 was a feature of the majority of the T cells. Analysis of IL-4 by cross-linkage of 125I-labeled IL-4 to IL-4 receptor with disuccinimidyl suberate indicated that the IL-4-IL-4 receptor complex was the same size in the resting and up-regulated cells, implying that the same receptor species found in resting cells was up-regulated in response to IL-4.

=> dis ibib abs 16 90-99

ANSWER 90 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on L6 STN

ACCESSION NUMBER: 1999:131347 BIOSIS DOCUMENT NUMBER: PREV199900131347

TITLE:

Peptides as drugs.

AUTHOR(S):

SOURCE:

Edwards, C. M. B.; Cohen, M. A.; Bloom, S. R.

CORPORATE SOURCE:

ICSM Endocrine Unit, Hammersmith Hosp., London, UK QJM, (Jan., 1999) Vol. 92, No. 1, pp. 1-4. print.

CODEN: QJMEA7. ISSN: 0033-5622.

DOCUMENT TYPE:

Article Editorial

LANGUAGE:

English

ENTRY DATE:

Entered STN: 17 Mar 1999

Last Updated on STN: 17 Mar 1999

L6 ANSWER 91 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER:

1998:706077 CAPLUS

DOCUMENT NUMBER:

129:321206

TITLE:

Sustained-release alginate gels

INVENTOR(S):

Goldenberg, Merrill Seymour; Beekman, Alice C.

PATENT ASSIGNEE(S):

Amgen Inc., USA

SOURCE:

LANGUAGE:

PCT Int. Appl., 41 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PAT	PATENT NO.				KINI)	DATE				LICAT				Ε	ATE	
WO	9846	211			A1	-	1998	1022	,		1998-1				1	.9980	414
	W:	AL,	AM,	AT,	AU,	AZ,	BA,	BB,	BG,	BR	, BY,	CA,	CH,	CN,	CU,	CZ,	DE,
		DK,	EE,	ES,	FI,	GB,	GE,	GH,	GM,	GW,	, HU,	ID,	IL,	IS,	JP,	KE,	KG,
		KP,	KR,	KZ,	LC,	LK,	LR,	LS,	LT,	LU,	, LV,	MD,	MG,	MK,	MN,	MW,	MX,
		NO,	NZ,	PL,	PT,	RO,	RU,	SD,	SE,	SG	, SI,	SK,	SL,	TJ,	TM,	TR,	TT,
		UA,	UG,	UZ,	VN,	YU,	ZW,	AM,	AZ,	BY,	, KG,	KZ,	MD,	RU,	TJ,	TM	
	RW:	GH,	GM,	KE,	LS,	MW,	SD,	SZ,	ŪĠ,	ZW,	, AT,	BE,	CH,	CY,	DE,	DK,	ES,
		FI,	FR,	GB,	GR,	IE,	IT,	LU,	MC,	NL	, PT,	SE,	BF,	ВJ,	CF,	CG,	CI,
		CM,	GA,	GN,	ML,	MR,	NE,	SN,	TD,	TG							
US	2002	0016	19		A1		2002	0103		US :	1997-	8427	56		1	9970	417
	6656																
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	2286																
											1998-				•		
EP	9753.	33			A1		2000	0202		EP :	1998-	9155	92		1	.9980	414
	R:	AT,	BE,	CH,	DE,	DK,	ES,	FR,	GB,	GR,	, IT,	LI,	LU,	NL,	SE,	MC,	PT,
					LV,								٠				
											1998-					.9980	
TW	5777	55			В		2004	0301		TW :	1998-	8710	5641		1	9980	414
•					A		2000	0630			1999-						
RIORITY	Y APP	LN.	INFO	. :							1997-						
						_					1998-1					.9980	

AB The present invention relates to sustained-release formulations using alginate gel beads. Small alginate beads were prepared by using 25mM ZnCl2 in the bath. As the concentration of the leptin in the bead increased, the fractional release of leptin from the bead decreased.

REFERENCE COUNT:

THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 92 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

3

ACCESSION NUMBER:

1998:323165 CAPLUS

DOCUMENT NUMBER:

129:8577

TITLE: INVENTOR(S): Methods for regulating angiogenesis Isner, Jeffrey M.; Asahara, Takayuki

PATENT ASSIGNEE(S):

St. Elizabeth's Medical Center of Boston, Inc., USA

SOURCE:

PCT Int. Appl., 57 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

LANGUAGE:

Patent English

2

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PATENT NO.		KIND	DATE	APPLICATION NO.	DATE
WO 9819712		A1	19980514	WO 1997-US19935	19971106
W: AU, C	CA, JP				
RW: AT, E	BE, CH,	DE, DK	, ES, FI,	FR, GB, GR, IE, IT, LU	, MC, NL, PT, SE
US 5980887		A	19991109	US 1996-744882	19961108
CA 2271690		A1	19980514	CA 1997-2271690	19971106
AU 9852432		A	19980529	AU 1998-52432	. 19971106
AU 743267		B2	20020124		
EP 941125		Al	19990915	EP 1997-947319	19971106
R: DE, E	FR, GB,	IT			
JP 2001503427	7	T	20010313	JP 1998-521645	19971106

EP 2005-17496 19971106 EP 1618898 A2 20060125

R: DE, FR, GB, IT

PRIORITY APPLN. INFO.: A 19961108 US 1996-744882 EP 1997-947319 A3 19971106

> WO 1997-US19935 W 19971106

In accordance with the present invention, EC (endothelial cell) AB progenitors can be used in a method for regulating angiogenesis, i.e., enhancing or inhibiting blood vessel formation, in a selected patient and in some preferred embodiments for targetting specific locations. For example, the EC progenitors can be used to enhance angiogenesis or to deliver an angiogenesis modulator, e.g. anti- or pro-angiogenic agents, resp. to sites of pathol. or utilitarian angiogenesis. Addnl., in another embodiment, EC progenitors can be used to induce reendothelialization of an injured blood vessel, and thus reduce restenosis by indirectly inhibiting smooth muscle cell proliferation.

THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS REFERENCE COUNT: 11

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ANSWER 93 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6 reserved on STN

1998320520 EMBASE ACCESSION NUMBER:

Successful treatment of a Jehovah's witness with acute TITLE:

promyelocytic leukemia by all-trans retinoic acid.

Kajiquchi T.; Yamamoto Y.; Miyata Y.; Saito M.; Takeyama H. **AUTHOR:**

Dr. T. Kajiguchi, Department of Internal Medicine, Nagoya CORPORATE SOURCE:

Ekisaikai Hospital, 4-66 Shonen-cho, Nakagawa-ku, Nagoya

454-8502, Japan

Biotherapy, (1998) Vol. 12, No. 8, pp. 1159-1163. . SOURCE:

Refs: 7

ISSN: 0914-2223 CODEN: BITPE

COUNTRY: Japan

Journal; Article DOCUMENT TYPE:

General Pathology and Pathological Anatomy 005 FILE SEGMENT:

> Internal Medicine 006

Cancer 016 Hematology 025

037 Drug Literature Index

Japanese LANGUAGE:

English; Japanese SUMMARY LANGUAGE:

Entered STN: 15 Oct 1998 ENTRY DATE:

Last Updated on STN: 15 Oct 1998

A 23-year-old woman was referred to our hospital for treatment of acute AB promyelocytic leukemia (APL). She was a Jehovah's Witness and would not accept blood products. The Ethics Committee of our hospital recommended accommodation of her religious beliefs, because the patient and her family thoroughly understood and accepted the increased risk of fatal bleeding and severe hypoxia due to anemia during the therapy without using blood products. After the patient signed a special consent form, we began treatment with all- trans retinoic acid (ATRA, 45 mg/m2 daily). She also received erythropoietin and granulocyto colony-stimulating factor. Sixty days after administration of ATRA, she achieved a complete remission (CR). After CR she received 3 cycles of chemotherapy for consolidation, which is according to the protocol of the Japan Adult Leukemia Study Group (AML-92). During the therapy, the patient did not receive blood products. No PML-RARA amplification products were detected by the reverse transcriptase polymerase chain reaction in her bone marrow at the end of the chemotherapy Blood product support is usually requisite for standard chemotherapy for APL because of the bleeding tendency. ATRA can induce differentiation of the leukemic clone without myelosuppression, so we can treat APL patients without using blood products.

ANSWER 94 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6 reserved on STN

1998359165 EMBASE ACCESSION NUMBER:

Strategies for the use of epoetin alfa in breast cancer TITLE:

patients.

Del Mastro L.; Venturini M. **AUTHOR:**

Dr. L. Del Mastro, Oncologia Medica 1, Isto. Nazionale CORPORATE SOURCE:

Ricerca Cancro, L. go Rosanna Benzi 10, 16132 Genova,

Italy. mventur@hp380.ist.unige.it

Oncologist, (1998) Vol. 3, No. 5, pp. 314-318. . SOURCE:

Refs: 27

ISSN: 1083-7159 CODEN: OCOLF6

COUNTRY: United States DOCUMENT TYPE: Journal; Article FILE SEGMENT: 016 Cancer Hematology 025 Pharmacology 030

> 037 Drug Literature Index Adverse Reactions Titles 038

English LANGUAGE: SUMMARY LANGUAGE: English

Entered STN: 19 Nov 1998 ENTRY DATE:

Last Updated on STN: 19 Nov 1998

Anemia is a common complication in cancer patients undergoing AB chemotherapy, and its severity depends on both the type of antineoplastic drugs and the clinical status of the patient. Breast cancer patients undergoing standard chemotherapy develop clinically significant anemia in up to 25% of cases. This percentage, moreover, increases up to 63% when more intensive chemotherapy regimens are used. The therapeutic use of erythropoietin in anemic patients, i.e., in patients with hemoglobin levels below 9-10.5 g/dl, is able to correct the anemic status in nearly 40%-80% of such patients, but it does not completely eliminate the need of blood transfusions: 20%-40% of patients need to be transfused despite the erythropoietin treatment. An alternative strategy for optimizing the erythropoietin treatment is its use in the prevention of anemia, i.e., in patients with normal hemoglobin values but at high risk of becoming anemic. In a phase III study, we evaluated the role of erythropoietin in the prevention of anemia in breast cancer patients undergoing dose-intensive chemotherapy. Clinically significant anemia occurred in 52% (95% CI = 33-69) of control patients and in no patient (95% CI = 0-14) in the erythopoietin arm (p = .00001). After six cycles of chemotherapy the mean hemoglobin decrease was 3.05 g/dl (± 1.0, 95% CI = 2.6-3.5) in the control arm and 0.8 g/dl (± 1.4, 95% CI = 0.3-1.4) in the erythropoietin arm. Moreover, 6.4% of control patients needed blood transfusion compared to no patients in the erythropoietin arm. Erythropoietin is active in both the treatment and the prevention of anemia in cancer patients undergoing chemotherapy. Due to its high economic cost, efforts should be made to identify subsets of patients in whom the preventive use could be cost-effective. Patients undergoing chemotherapy associated with a high risk of anemia could benefit from preventive use of erythropoietin in special circumstances, such as presence of risk of myocardial or cerebral ischemia, uncommon blood group, or religious beliefs hindering blood transfusions. Moreover, anemia prevention could be considered in patients at high risk of requiring blood transfusions, such as patients with low baseline value of hemoglobin or with a hemoglobin decrease of ≥2 g/dl after the first cycle of chemotherapy.

ANSWER 95 OF 113 MEDLINE on STN DUPLICATE 9 L6

1998140501 MEDLINE ACCESSION NUMBER: PubMed ID: 9479872 DOCUMENT NUMBER:

Markedly high plasma erythropoietin and TITLE:

granulocyte-colony stimulating

factor levels in patients with paroxysmal nocturnal

hemoglobinuria.

Nakakuma H; Nagakura S; Kawaguchi T; Horikawa K; Iwamoto N; AUTHOR:

Kagimoto T; Takatsuki K

Second Department of Internal Medicine, Kumamoto University CORPORATE SOURCE:

School of Medicine, Japan.

International journal of hematology, (1997 Dec) Vol. 66, SOURCE:

No. 4, pp. 451-7.

Journal code: 9111627. ISSN: 0925-5710.

PUB. COUNTRY: Ireland

Journal; Article; (JOURNAL ARTICLE) DOCUMENT TYPE:

English LANGUAGE:

Priority Journals FILE SEGMENT:

ENTRY MONTH:

199803

ENTRY DATE:

Entered STN: 26 Mar 1998

Last Updated on STN: 26 Mar 1998 Entered Medline: 17 Mar 1998

In patients with paroxysmal nocturnal hemoglobinuria (PNH), we measured ABplasma concentrations of endogenous hematopoiesis-regulatory cytokines to characterize bone marrow (BM) hypoplasia which is a major cause of death.

Contrary to 10 healthy individuals, all 14 patients with PNH showed

increases of erythropoietin (Epo) and granulocyte-

colony stimulating factor (G-CSF). There were

no signs of infection, renal dysfunction or hypoxia. The lower

the hemoglobin level and granulocyte count, the higher the plasma Epo and G-CSF levels. In contrast, marked differences were not found in the levels of interleukin-3 (IL-3), tumor necrosis factor-alpha (TNF-alpha),

stem cell factor (SCF), granulocyte/macrophage-

colony stimulating factor (GM-CSF), or

interferon-gamma) (IF-gamma). The cytokine profiles of PNH patients were quite similar to those of patients with aplastic anemia (AA) and myelodysplastic syndrome (MDS). The cytokine profiles may support a pathological relationship between PNH and these stem cell disorders.

ANSWER 96 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on L6 STN

1997:199747 BIOSIS ACCESSION NUMBER:

DOCUMENT NUMBER:

PREV199799498950

TITLE:

Hematology of the elderly patient: The red series: Anemia

of the elderly patient.

AUTHOR(S):

Florez-Tascon Sixto, F. J.; Sanchez-Escribano, F.; Siguin

Gomez, A.; Herraez, R.; Cobos, J.; Ruiz Martin, J. Geriatrika (Madrid), (1997) Vol. 13, No. 1, pp. 17-21.

SOURCE: ISSN: 0212-9744.

DOCUMENT TYPE:

Article

LANGUAGE:

Spanish

ENTRY DATE:

Entered STN: 12 May 1997

Last Updated on STN: 12 May 1997

ANSWER 97 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6

reserved on STN

ACCESSION NUMBER:

97330591 EMBASE

DOCUMENT NUMBER:

1997330591

TITLE:

[Erythropoietin: Biochemical profile, biological records, indications and therapeutic results in

hematology].

ERITROPOIETINA: PROFILO BIOCHIMICO, RICORDI BIOLOGICI, INDICAZIONI E RISULTATI TERAPEUTICI IN EMATOLOGIA.

AUTHOR:

Marmont A.M.

CORPORATE SOURCE:

Prof. A.M. Marmont, II Divisione di Ematologia, Ospedale San Martino, Piazzale R. Benzi 10, 16132 Genova, Italy Tumori, (1997) Vol. 83, No. 4 SUPPL. 2, pp. S3-S15. .

SOURCE:

Refs: 146

ISSN: 0300-8916 CODEN: TUMOAB

COUNTRY:

Italy

DOCUMENT TYPE:

Journal; Conference Article

FILE SEGMENT:

Cancer 016

Hematology 025

029 Clinical Biochemistry 037 Drug Literature Index

LANGUAGE: Italian SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 1 Dec 1997

Last Updated on STN: 1 Dec 1997

This review has two objects: a brief recapitulation of the biological ABbackground of erythropoietin (EPO), and a review of its clinical utilization in hematology. EPO, both in its naturally occurring and recombinant form (rH-EPO), is a single chain glycoprotein with an approximate molecular weight of 30,000 to 34,000 kD. glycosilation is essential for its activity in vivo, since asialoEPO is readily cleared by the hepatic asialoglycoprotein receptor. This impedes the recombinant molecule's synthesis in biologic cultures other than mammalian cells (Chinese hamster's ovary cells), and inevitably increases costs. If in vitro glycosilation of E. coli-derived rH-EPO could be achieved, the clinical utilization of the product would be considerably enhanced, most especially when very high doses are necessary, as discussed later. There is no antigenic diversity between natural and recombinant EPO, so that out of the enormous clinical experience only one single case of immunization has been recorded. Almost paradoxically there are however three published cases of pure red cell aplasia (PRCA) caused by immunization against autologous EPO. It is now established that in adults EPO is synthetized in renal peritubular interstitial cells, although some residual activity remains in the liver. Hypoxia results in a rapid induction of EPO expression, although the role of the oxygen sensor system is still debated. Cellular targets are notoriously erythroid progenitors and precursors (BFU-E, CFU-E, early and intermediate erythroblasts). The global erythropoietic activity resulted in various effects (proliferation, differentiation, survival), but most probably each single effect is integrated with and complementary of the others. The utilization of rH-EPO in hematologic diseases came much later than its dramatic success in renal anemia. A variety of tools useful for assessing the possible beneficial effects of rH-EPO in clinical hematology has been proposed, among which a low level of endogenous EPO is a good predictor for therapeutic success. 'Hemopathic' anemia can be subdivided into three categories: patients with normal erythropoiesis due to inadequate EPO production (anemia of prematurity), patients with depressed but nonclonal erythropoiesis (chemotherapy, lymphoid malignancies such as multiple myeloma MM and chronic lymphatic leukemia - CCL) and patients with at least partially clonal anemia, such as paroxysmal nocturnal hemoglobinuria (PNH), hemoglobinopathies, myelodysplastic syndromes (MDS) and others. Results in the first category of patients are, as expected, prompt and satisfactory with physiologic doses. Although therapeutic strategy for MM is moving fast to curative intents, the utilization of rH-EPO is indicated or the control of anemia in conservatively-treated patients. In the third category the most important and controversial area is MDS. Significant erythropoietic results are generally obtained in about 20% of patients; however, the association with G-CSF has considerably enhanced the response rate. In the field of bone marrow transplantation there is an inadequate production of endogenous EPO in the allogeneic setting, and randomized studies have shown the benefits of rH-EPO in this situation. However, the most important results have been and are obtained in post-major-ABO incompatible PRCA, when the removal of the recipient's isohemagglutinins does not resolve the anemia. High and very high doses of rH-EPO (even over 500 Ul/kg/day for 2-4 weeks) may resolve this occasionally quite refractory condition. Although extremely expensive, this treatment may be life-saving when an otherwise successful allogeneic transplant is at the risk of failure because of this relatively uncommon but severe immunohematologic complication.

L6 ANSWER 98 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1996:756546 CAPLUS

DOCUMENT NUMBER: 126:17804

Human antibodies derived from immunized xenomice TITLE: Kucherlapati, Raju; Jakobovits, Aya; Klapholz, Sue; INVENTOR(S):

Brenner, Daniel G.; Capon, Daniel J.

Cell Genesys, Inc., USA PATENT ASSIGNEE(S): PCT Int. Appl., 64 pp. SOURCE:

CODEN: PIXXD2

DOCUMENT TYPE: Patent LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO. KIND DATE APPLICATION NO. DATE _ _ _ _ WO 1995-US5500 A1 19961031 WO 9634096 19950428 W: AU, CA, FI, HU, JP, KR, NO, NZ RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE CA 1995-2219486 19961031 19950428 CA 2219486 A1 19961118 AU 9524668 Α AU 1995-24668 19950428 EP 823941 19980218 EP 1995-918935 A1 19950428 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE JP 11505107 T 19990518 JP 1995-532463 19950428 PRIORITY APPLN. INFO.: WO 1995-US5500 W 19950428 Antibodies with fully human variable regions against a specific antigen AB can be prepared by administering the antigen to a transgenic animal which has been modified to produce such antibodies in response to antigenic challenge, but whose endogenous loci have been disabled. Various subsequent manipulations can be performed to obtain either antibodies per

ANSWER 99 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6

reserved on STN

ACCESSION NUMBER: 96228211 EMBASE

se or analogs thereof.

1996228211 DOCUMENT NUMBER:

Bibtech finds a growth industry. TITLE:

Roush W. AUTHOR:

Science, (1996) Vol. 273, No. 5273, pp. 300-301. . SOURCE:

ISSN: 0036-8075 CODEN: SCIEAS

United States COUNTRY: Journal: Note DOCUMENT, TYPE:

Biophysics, Bioengineering and Medical FILE SEGMENT: 027

> Instrumentation Pharmacology

037 Drug Literature Index

English LANGUAGE:

Entered STN: 28 Oct 1996 ENTRY DATE:

030

Last Updated on STN: 28 Oct 1996

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

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ANSWER 80 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6

reserved on STN

2000337455 EMBASE ACCESSION NUMBER:

Treatment of leukemia, lymphoma and cancer - 13th TITLE:

International Symposium: Molecular Biology of

Hematopoiesis: 14-18 July 2000, New York, NY, USA.

Rose-John S. AUTHOR:

S. Rose-John, Department of Biochemistry, CORPORATE SOURCE:

Christian-Albrechts Universitat Kiel, Olshausenstrasse 40,

D-24098 Kiel, Germany. rosejohn@biochem.uni-kiel.de Current Opinion in Oncologic, Endocrine and Metabolic

SOURCE: Investigational Drugs, (2000) Vol. 2, No. 4, pp. 423-425. .

ISSN: 1464-8466 CODEN: COODF2

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Conference Article FILE SEGMENT: 037 Drug Literature Index

016 Cancer 025 Hematology 022 Human Genetics 030 Pharmacology

026 Immunology, Serology and Transplantation

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 13 Oct 2000

Last Updated on STN: 13 Oct 2000

This conference included sessions covering the clinical aspects of AB methodological CD34+ cell expansion, identification of the true hematopoietic stem cells, cancer and coagulation therapies, pathology of anemia in cancer patients following chemotherapy, molecular biology of erythropoietin signaling, and the role of hypoxia as a regulator of malignant cell growth. Sessions on lymphopoiesis, dendritic cells and the control of the immune response were presented in addition to updates on the JAK/STAT signaling pathways of cytokine receptors and on the construction and use of novel designer cytokines in the expansion of hematopoietic progenitor cells and gene therapy of human cancer. Innovation and new strategies for bone marrow transplantation and organ transplants were discussed, and the use of p53 gene transfer in the induction of apoptosis in malignant cells was evaluated. More than 400 scientific contributions were presented as plenary talks, short communications and poster presentations; approximately 250 to 300 people attended.

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ACCESSION NUMBER: 2000178806 EMBASE

TITLE: Applications of developmental biology to medicine and

animal agriculture.

AUTHOR: Smith R.C.; Rhodes S.J.

CORPORATE SOURCE: Dr. R.C. Smith, Department of Biology, IUPUI, 723 W.

Michigan Street, Indianapolis, IN 46202-5132, United States

SOURCE: Progress in Drug Research, (2000) Vol. 54, pp. 213-256.

Refs: 221

ISSN: 0071-786X CODEN: FAZMAE

COUNTRY: Switzerland

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 021 Developmental Biology and Teratology

Human GeneticsPharmacology

037 Drug Literature Index

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 8 Jun 2000

Last Updated on STN: 8 Jun 2000

AB With the complete sequence of the human genome expected by winter 2001, genomic-based drug discovery efforts of the pharmaceutical industry are focusing on finding the relatively few therapeutically useful genes from among the total gene set. Methods to rapidly elucidate gene function will have increasing value in these investigations. The use of model organisms in functional genomics has begun to be recognized and exploited and is one example of the emerging use of the tools of developmental biology in recent drug discovery efforts. The use of protein products expressed during embryogenesis and the use of certain pluripotent cell populations (stem cells) as candidate therapeutics are other applications of developmental biology to the treatment of human diseases. These agents may be used to repair damaged or diseased tissues by inducing or directing developmental programs that recapitulate embryonic processes to replace specialized cells. The activation or silencing of embryonic genes in the

disease state, particularly those encoding transcription factors, is another avenue of exploitation. Finally, the direct drug-induced manipulation of embryonic development is a unique application of developmental biology in animal agriculture.

L6 ANSWER 82 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000120188 EMBASE

TITLE: Prognostic significance of anemia and role of

. erythropoietin in radiation therapy.

AUTHOR: Smaniotto D.; Luzi S.; Morganti A.G.; Cellini N.

CORPORATE SOURCE: Dr. D. Smaniotto, Istituto di Radiologia, Universita

Cattolica del Sacro Cuore, Policlinico A. Gemelli, largo A. Gemelli 8, 00168 Roma, Italy. radioterapia2.3e@rm.unicatt.i

t

SOURCE: Tumori, (2000) Vol. 86, No. 1, pp. 17-23. .

Refs: 36

ISSN: 0300-8916 CODEN: TUMOAB

COUNTRY: Italy

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 014 Radiology 016 Cancer

037 Drug Literature Index

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 13 Apr 2000

Last Updated on STN: 13 Apr 2000

Anemia represents a common finding in cancer patients, especially at an AB advanced stage. Anemia has an impact on the quality of life and at the same time seems to markedly limit the disease control that can be achieved The results of a series of clinical studies published with radiotherapy. in the last decade allow some general observations: 1. the administration of erythropoietin, especially if associated to ferrous sulfate is able to increase hemoglobulin levels in cancer patients undergoing radiation therapy (combined with concomitant chemotherapy); 2. erythropoietin stimulation of hemoglobin in anemia decreases the need for blood transfusion in cancer patients; 3. tumor response to radiation therapy appears to be enhanced by erythropoietin -induced hemoglobulin increase. Further clinical studies are required for assessment of indications, identification of optimal administration modalities, cost-analysis of this promising therapy for patients undergoing radiation therapy.

L6 ANSWER 83 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000081357 EMBASE

TITLE: Manifestations and treatment of Schimke immuno-osseous

dysplasia: 14 new cases and a review of the literature.

AUTHOR: Boerkoel C.F.; O'Neill S.; Andre J.L.; Benke P.J.;

Bogdanovic R.; Bulla M.; Burguet A.; Cockfield S.; Cordeiro I.; Ehrich J.H.H.; Frund S.; Geary D.F.; Ieshima A.; Illies F.; Joseph M.W.; Kaitila I.; Lama G.; Leheup B.; Ludman M.D.; McLeod D.R.; Medeira A.; Milford D.V.; Ormala T.; Rener-Primec Z.; Santava A.; Santos H.G.; Schmidt B.; Smith

G.C.; Spranger J.; Zupancic N.; Weksberg R.

CORPORATE SOURCE: R. Weksberg, Hospital for Sick Children, Div. of

Clinic./Metabolic Genetics, University of Toronto, 555

University Avenue, Toronto, Ont. M5G 1X8, Canada.

rweksbg@sickkids.on.ca

SOURCE: European Journal of Pediatrics, (2000) Vol. 159, No. 1-2,

pp. 1-7. . Refs: 23

ISSN: 0340-6199 CODEN: EJPEDT

COUNTRY: Germany

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 007 Pediatrics and Pediatric Surgery

022 Human Genetics

037 Drug Literature Index

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 16 Mar 2000

Last Updated on STN: 16 Mar 2000

Schimke immuno-osseous dysplasia (SIOD) is a rare autosomal recessive AB spondylo-epiphyseal dysplasia. The characteristic features of SIOD include 1) short stature with hyperpigmented macules and an unusual facies, 2) proteinuria with progressive renal failure, 3) lymphopenia with recurrent infections, and 4) cerebral ischaemia. Although 25 patients have been reported with this disorder, the clinical course and phenotype of SIOD are not well characterized. This report summarizes the clinical findings, course and treatment of reported patients and includes 14 additional patients with SIOD. We emphasize the high incidence of cerebral ischaemia and ocular abnormalities, define the high incidence of thyroid dysfunction and blood cytopenia, and confirm the absence of effective and durable medical therapies. Conclusion: Schimke immuno-osseous dysplasia is a multi-system autosomal recessive disorder with variable expression that affects the skeletal, renal, immune, vascular, and haematopoietic systems. Medical therapy is limited especially for more severely affected individuals.

L6 ANSWER 84 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1999:795994 CAPLUS

DOCUMENT NUMBER: 132:31744

TITLE: Gene probes used for genetic profiling in healthcare

A DOT TOARTON NO

screening and planning

INVENTOR(S): Roberts, Gareth Wyn
PATENT ASSIGNEE(S): Genostic Pharma Ltd., UK

PATENT ASSIGNEE(S): Genostic Pharma Ltd., UK SOURCE: PCT Int. Appl., 745 pp.

DOCUMENT TYPE: CODEN: PIXXD2
Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

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PATENT NO.			KINI		DATE		Ì	APPL:	ICAT:	ION I	NO.	· 	D	ATE	·		
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		DE,	DK,	EE,	ES,	FI,	GB,	GD,	GE,	GH,	GM,	HR,	HU,	ID,	IL,	IN,	IS,
		JP,	KE,	KG,	KΡ,	KR,	KZ,	LC,	LK,	LR,	LS,	LT,	LU,	LV,	MD,	MG,	MK,
		MN,	MW,	MX,	NO,	NZ,	PL,	PT,	RO,	RU,	SD,	SE,	SG,	SI,	SK,	SL,	TJ,
		TM,	TR,	TT,	UA,	UG,	US,	UZ,	VN,	YU,	ZA,	ZW,	AM,	AZ,	BY,	KG,	KZ,
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A 19980808 GB 1998-17200 A 19980814 GB 1998-17632 GB 1998-17943 A 19980819

There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

CAPLUS COPYRIGHT 2007 ACS on STN ANSWER 85 OF 113 L6

ACCESSION NUMBER:

1999:795993 CAPLUS

DOCUMENT NUMBER:

132:31743

TITLE:

AB

Gene probes used for genetic profiling in healthcare

screening and planning

INVENTOR(S):

Roberts, Gareth Wyn Genostic Pharma Limited, UK

PATENT ASSIGNEE(S): SOURCE:

PCT Int. Appl., 149 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PAI	CENT 1	NO.			KINI	Ο.	DATE			APPL:	ICAT:	ION I	NO.	DATE			
WO	9964	626			A2	•	1999	1216	1	WO 1	999-(GB17	79		. 19	9990	504
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		JP,	KE,	KG,	KP,	KR,	KZ,	LC,	LK,	LR,	LS,	LT,	LU,	LV,	MD,	MG,	MK,
		MN,	MW,	MX,	NO,	NZ,	PL,	PT,	RO,	RU,	SD,	SE,	SG,	SI,	SK,	SL,	TJ,
		TM,	TR,	TT,	UA,	UG,	US,	UZ,	VN,	ΥU,	ZA,	ZW,	AM,	AZ,	BY,	KG,	KZ,
		MD,	RU,	TJ,	TM												
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		ES,	FI,	FR,	GB,	GR,	IE,	IT,	LU,	MC;	NL,	PT,	SE,	BF,	ВJ,	CF,	CG,
•		CI,	CM,	GA,	GN,	GW,	ML,	MR,	NE,	SN,	TD,	TG					
CA	2330	929			A1		1999	1216		CA 1	999-	2330	929		1	9990	604
AU	9941	586			Α		1999	1230		AU 1	999-	4158	6		1	9990	604
ΑU	7665	44			B2		2003	1016									
AU	9941	587			A		1999	1230	• •	AU 1:	999-	4158	7		1	9990	604
GB	2339	200			Α		2000	0119		GB 1	999-	1291	4		1	9990	604
GB	2339	200			В		2001	0912									

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20010321
                                           EP 1999-925207
                                                                  19990604
    EP 1084273
                         A1
        R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, FI
    JP 2003528564
                               20030930
                                           JP 2000-553616
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                               20031023
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                                           US 2002-206568
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PRIORITY APPLN. INFO.:
                                           GB 1998-28289
                                                               A 19981223
                                           GB 1998-16086
                                                               A 19980724
                                           GB 1998-16921
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                                           GB 1998-17097
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                                                               A 19980808
                                           GB 1998-17200
                                           GB 1998-17632
                                                               A 19980814
                                           GB 1998-17943
                                                               A 19980819
                                           US 1999-325123
                                                               B1 19990603
                                                               W 19990604
                                           WO 1999-GB1779
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There is considerable evidence that significant factor underlying the ABindividual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

CAPLUS COPYRIGHT 2007 ACS on STN ANSWER 86 OF 113 L6

ACCESSION NUMBER:

1999:594848 CAPLUS

DOCUMENT NUMBER:

131:223977

TITLE:

Compositions and methods for inducing

neovascularization using a vascularization modulating

agent such as GM-CSF

INVENTOR(S): PATENT ASSIGNEE(S): Isner, Jeffrey M.; Asahara, Takayuki St. Elizabeth's Medical Center, USA

SOURCE:

PCT Int. Appl., 74 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

'PAT	TENT 1	NO.			KIN	D	DATE		APPLICATION NO.						DA	ATE	
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Μ̈́O	9945	775			Al		1999	0916	1	WO 1	999-1	JS51:	3 0		19	9990	309
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		DK,	EE,	ES,	FI,	GB,	GE,	GH,	GM,	HR,	HU,	ID,	IL,	IS,	JP,	KE,	KG,
		KP,	KR,	ΚZ,	LC,	LK,	LR,	LS,	LT,	LU,	LV,	MD,	MG,	MK,	MN,	MW,	MX,
		NO,	NZ,	PL,	PT,	RO,	RU,	SD,	SE,	SG,	SI,	SK,	SL,	TJ,	TM,	TR,	TT,
		UA,	UG,	US,	UZ,	VN,	YU,	ZW,	AM,	AZ,	BY,	KG,	ΚZ,	MD,	RU,	TJ,	TM
	RW:	GH,	GM,	KE,	LS,	MW,	SD,	SL,	SZ,	UG,	ZW,	AT,	BE,	CH,	CY,	DE,	DK,
		ES,	FI,	FR,	GB,	GR,	IE,	IT,	LU,	MC,	NL,	PT,	SE,	BF,	ВJ,	CF,	CG,
		CI,	CM,	GA,	GN,	GW,	ML,	MR,	NE,	SN,	TD,	TG					
CA	2322	559			Al		1999	0916	(CA 1	999-2	2322	559		19	9990	309
AU	9930	737			Al 19990916 CA 1999-2322559 A 19990927 AU 1999-30737						19	9990	309				
AU	7662	38			B2		2003	1009									
EP	1061	800			Al	A1 20001227				EP 1	999-	9123	44		19	9990	309
	R:	AT,	BE,	CH,	DE,	.DK,	ES,	FR,	GB,	GR,	IT,	LI,	LU,	NL,	SE,	MC,	PT,

IE, FI

20020226 JP 2000-535201 19990309 JP 2002506008 US 2003-714574 A1 20041118 US 2004228835 20031114 US 1998-77262P P 19980309 PRIORITY APPLN. INFO.: US 1999-265041 A3 19990309 WO 1999-US5130 W 19990309 US 2000-698323 A1 20001027

The present invention generally provides methods for modulating formation AB of new blood vessels. In one embodiment, the methods include administering to a mammal an effective amount of a vascularization modulating agent (such as granulocyte macrophagecolony stimulating factor) sufficient to form the new blood vessels. Addnl. provided are methods for preventing or reducing the severity of blood vessel damage in a mammal which methods preferably include administering to the mammal an effective amount of GM-CSF or another vascularization modulating agent. Instead of the proteins themselves being administered, the DNA encoding for the vascularization modulating agents can be administered. Addnl., the vascularization modulating agent can also be coadministered with at least one angiogenic protein. In addition to administering the vascularization modulating agent to treat ischemic tissue, it's also possible to contact isolated endothelial progenitor cells (EPCs) with an amount of an angiogenic protein sufficient to induce proliferation of the EPCs and then administer the proliferated EPCs to treat the ischemic tissue. Provided also as part of this invention are pharmaceutical products and kits for inducing formation of new blood vessels in the mammal.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 87 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1999302671 EMBASE

TITLE: [Efficiency, safety and tolerability of recombinant human

Interleukin-3 (rhIL-3) as supportive treatment under dose-intensified Carboplatin- chemotherapy in ovarian cancer patients with special regard to thrombocytopenia). EFFIZIENZ, SICHERHEIT UND VERTRAGLICHKEIT VON REKOMBINANTEM

HUMANEN INTERLEUKIN-3 (RHIL-3) ALS SUPPORTIVE THERAPIE BEGLEITEND ZUR DOSISINTENSIVIERTEN

CARBOPLATIN-HALTIGEN CHEMOTHERAPIE BEI PATIENTINNEN MIT OVARIALKARZINOM UNTER BESONDERER BERUCKSICHTIGUNG DER

THROMBOZYTOPENIE.

AUTHOR: Meden H.; Fock M.; Krauss T.; Kuhn W.

CORPORATE SOURCE: Dr. H. Meden, Universitats-Frauenklinik Gottingen,

Robert-Koch-Strasse 40, D-37075 Gottingen, Germany.

hmeden@med.uni-goettingen.de

SOURCE: Zentralblatt fur Gynakologie, (1999) Vol. 121, No. 8, pp.

375-383. . Refs: 20

ISSN: 0044-4197 CODEN: ZEGYAX

COUNTRY: Germany

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 010 Obstetrics and Gynecology

016 Cancer

026 Immunology, Serology and Transplantation

037 Drug Literature Index038 Adverse Reactions Titles

LANGUAGE: German

SUMMARY LANGUAGE: English; German

ENTRY DATE: Entered STN: 10 Sep 1999

Last Updated on STN: 10 Sep 1999

AB Objective: In a prospective, randomized, placebo-controlled double-blind trial we evaluated to what extent a dose-intensification of adjuvant chemotherapy is possible with the help of Interleukin-3 (rhIL-3).

Material and Methods: following initial surgery, 12 patients with primary ovarian cancer have been treated with Carboplatin and Cyclophosphamide (dosage: AUC 4 according to Calvert). After randomisation, a group of 6 patients prophylacticly received rhIL-3 against myelosuppression on days 3-12 of the cycle, in contrast to a group of 6 patients who received placebo-injections. Results: The patients treated with rhIL-3 showed less hematologic side- effects. Adherence to 4-weekly chemotherapy courses was more frequent in the rhIL-3-group (73 % vs. 44 %, p = 0.005). An intensification of the chemotherapy with 3-weekly courses did not succeed significantly. Observed side-effects of rhIL-3-therapy were headaches, fever, flu-like symptoms, rashes and blisters at the site of injection which excluded 2 of 6 patients from the study. Conclusions: Supportive rhIL-3 to adjuvant Carboplatin-based chemotherapy enables a better keeping of 4-weekly courses in contrast to the placebo-group due to faster recovery of hematologic parameters. Due to the side-effects, of IL-3, this cytokine cannot be recommended for routine clinical use.

L6 ANSWER 88 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1999248033 EMBASE

TITLE: Changes of cytokine mRNA in peripheral blood mononuclear

cells from unresectable non-small cell lung cancer patients

before and after clarithromycin therapy.

AUTHOR: Majima T.; Mikasa K.; Hamada K.; Konish M.; Maeda K.;

Sakamoto M.; Yoshimoto E.; Murakawa K.; Ueda K.; Kita E.;

Narita N.

CORPORATE SOURCE: T. Majima, Internal Medicine II, Nara Medical University,

840 Shijouchou, Kashihara, Nara 634, Japan

SOURCE: Japanese Journal of Chemotherapy, (1999) Vol. 47, No. 6,

pp. 345-348. . Refs: 13

ISSN: 1340-7007 CODEN: NKRZE5

COUNTRY:

Japan

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis

016 Cancer

037 Drug Literature Index

LANGUAGE: Japanese

SUMMARY LANGUAGE: English; Japanese

ENTRY DATE: Entered STN: 2 Aug 1999

Last Updated on STN: 2 Aug 1999

We have reported that long term clarithromycin (CAM) therapy improves the survival time of patients with non-small cell lung cancer. In the present study, we examined peripheral blood mononuclear cells for changes in cytokine mRNA by RT-PCR before and after CAM therapy. The study included 15 patients with unresectable non-small cell lung cancer. Before CAM therapy, 13 patients received basic therapy consisting of chemotherapy, radiotherapy or both. Two patients received no basic therapy. Interleukin-10 (IL-10), Interleukin-12 (II-12), Interferon-gamma (IFN- γ) mRNA were measured before and at one and three months after starting CAM therapy. IL-12 and IFN- γ mRNA were significantly increased, and IL-10 m RNA was decreased. The results suggest that CAM exhibits an antitumor effect that promotes Th-lymphocytes shows a Th 1-like cytokine production.

L6 ANSWER 89 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2000:177684 CAPLUS

DOCUMENT NUMBER: 132:292537

TITLE: Human cytokines modulate arterial vascular tone via

endothelial receptors

AUTHOR(S): Iversen, Per Ole; Nicolaysen, Anne; Kvernebo, Knut;

Benestad, Haakon B.; Nicolaysen, Gunnar

CORPORATE SOURCE: Department of Physiology, Institute of Basic Medical Sciences, University of Oslo, Oslo, N-0317, Norway

SOURCE:

Pfluegers Archiv (1999), 439(1-2), 93-100

CODEN: PFLABK; ISSN: 0031-6768

PUBLISHER:

Springer-Verlag

DOCUMENT TYPE:

Journal

LANGUAGE:

English

Only a few cytokines have been tested for their possible role in AB modulating vascular function. Moreover, no direct effect of cytokines on vascular tone has yet been throughly studied. We therefore examined whether a wide range of well-defined cytokines could directly affect vascular tone in isolated human arterial and venous segments from various organs. We found that the cytokines stem cell factor (maximal response with 1 mM), granulocyte colony-stimulating factor (0.1 mM) and erythropoietin (1 mM) relaxed, while tumor necrosis factor α (0.1 mM), interleukin (IL) 6 (10 mM) and IL-10 (0.1 mM) induced contraction of arterial but not of venous segments. The cytokines (maximal concentration tested was 1 mM) IL-3, IL-5, IL-13, macrophage colony-stimulating factor and granulocyte-macrophage colony-stimulating factor had no apparent effects on either arterial or venous tone. These vascular effects were endothelium-dependent as denuded arteries did not respond to any cytokine, and inhibition of nitric oxide synthase or endothelin receptor A abrogated the cytokine-induced changes in vascular tone. With immunohistochem. we found receptors for the active cytokines on the arterial endothelium. conclusion, several cytokines may modulate arterial vascular tone via endothelium-dependent mechanisms. Therefore cytokines might significantly modify blood supply to inflamed or ischemic tissues with elevated local

REFERENCE COUNT:

THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE COST IN U.S. DOLLARS

concns. of cytokines.

SINCE FILE TOTAL SESSION ENTRY

FULL ESTIMATED COST

177.92 178.34

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

38

SINCE FILE TOTAL SESSION ENTRY

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ANSWER 70 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6 reserved on STN

ACCESSION NUMBER:

2003463279 EMBASE

TITLE:

Treatment of hepatitis C virus infection in the allograft.

AUTHOR:

Neuberger J.

CORPORATE SOURCE:

J. Neuberger, Liver Unit, Queen Elizabeth Hospital,

Birmingham B15 2TH, United Kingdom.

James.Neuberger@uhb.nhs.uk

SOURCE:

Liver Transplantation, (2003) Vol. 9, No. 11, pp.

S101-S108. .

Refs: 46

ISSN: 1527-6465 CODEN: LITRFO

COUNTRY:

United States Journal; Article 009 Surgery

DOCUMENT TYPE: FILE SEGMENT:

030

Pharmacology

Health Policy, Economics and Management 036

Drug Literature Index 037 038 Adverse Reactions Titles

048 Gastroenterology

LANGUAGE:

English English

SUMMARY LANGUAGE: ENTRY DATE:

Entered STN: 1 Dec 2003

Last Updated on STN: 1 Dec 2003

Key Points 1. Recurrence of hepatitis C virus (HCV) in the graft is AB associated with a reduced quality of life and worse graft survival. 2. Pretransplantation, the severity of HCV recurrence may be reduced by reducing the pretransplantation load, by avoiding the use of organs from older donors, and by reducing the ischemic times. The effect of split livers on recurrence rates is uncertain. 3. The optimal immunosuppression regime has not been established but a heavy induction regime and treatment for acute rejection are associated with more viral replication and more graft damage. 4. Presently, there is no convincing evidence for preemptive treatment of HCV. 5. There are many studies on the effect of interferon with and without ribavirin for the treatment of HCV hepatitis. However, few are prospective, randomized, and controlled. 6. The current best treatment is with pegylated interferon and ribavirin; the dose and duration of treatment need to be established. Side-effects of treatment are common and reduction/withdrawal is frequent, but the regime is cost-effective. 7. The role of newer treatments remains to be established.

ANSWER 71 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN L6

ACCESSION NUMBER:

2002:184917 CAPLUS

DOCUMENT NUMBER:

136:268103

TITLE:

Method for modifying synthetic proteins with branched water-soluble polymer to improve their biol. activity

or pharmacokinetic properties

INVENTOR(S):

Kochendoerfer, Gerd; Kent, Stephen B. H.; Botti; Paolo; Low, Donald W.; Bradburne, James A.; Chen,

Shiah-Yun; Cressman, Sonya; Hunter, Christie L.; Kent,

Stephen B. H.; Low, Donald W.; Wilken, Jill G.

PATENT ASSIGNEE(S):

SOURCE:

Gryphon Sciences, USA PCT Int. Appl., 245 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent English

LANGUAGE:

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PAT	TENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO	2002020033	· Al	20020314	WO 2001-US21930	20010712
	W: AL, AM,	AT, AU, AZ	Z, BA, BB,	BG, BR, BY, CA, CH,	CN, CU, CZ, DE,
	DK, EE,	ES, FI, GB	B, GE, GH,	HU, IL, IS, JP, KE,	KG, KP, KR, KZ,
	LC; LK,	LR, LS, LT	r, LU, LV,	MD, MG, MK, MN, MW,	MX, NO, NZ, PL,
	PT, RO,	RU, SD, SE	E, SG, SI,	SK, SL, TJ, TM, TR,	TT, UA, UG, US,
	UZ, VN,	YU, ZW			
	RW: GH, GM,	KE, LS, MW	W, MZ, SD,	SL, SZ, TZ, UG, ZW,	AT, BE, CH, CY,
	DE, DK,	ES, FI, FR	R, GB, GR,	IE, IT, LU, MC, NL,	PT, SE, TR, BF,
-	BJ, CF,	CG, CI, CM	M, GA, GN,	GW, ML, MR, NE, SN,	TD, TG
CA	2412278	A1	20020314	CA 2001-2412278	20010712
AU	2001073385	A 5	20020322	AU 2001-73385	20010712
BR	2001013579	BR 2001-13579	20010712		

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20030618
                                         EP 2001-952654
    EP 1318827
                        A1
                                                               20010712
        R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
                        T
                              20040318
    JP 2004508338
                                         JP 2002-524516
                                                               20010712
    US 2004115774
                                                               20030108
                        A1
                              20040617
                                         US 2003-332385
                       B2
    US 7118737
                              20061010
                      A
Al
    NO 2003001047
                              20030508
                                         NO 2003-1047
                                                               20030306
                              20061019
    US 2006233747
                                         US 2006-446419
                                                               20060605
                                         US 2000-231339P P 20000908
PRIORITY APPLN. INFO.:
                                                           P 20000929
                                         US 2000-236377P
                                         WO 2001-US21930
                                                            W 20010712
                                         US 2003-332385
                                                            A1 20030108
```

The present invention relates to methods and compns. for modifying ABpeptides, polypeptides and proteins with polymers, especially glyco-mimetic polymers, so as to improve their biol. activity or pharmacokinetic properties. The invention provides seven synthetic erythropoiesis stimulating proteins and four RANTES derivs. having one or more branched water-soluble polymers attached thereto. The invention further provides methods and uses for such polymer-modified peptides, polypeptides and proteins. The invention is particularly suitable for use in the synthesis of polymer-modified synthetic bioactive proteins (Figure 1D), and of pharmaceutical compns. that contain such proteins.

REFERENCE COUNT:

THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ANSWER 72 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6 reserved on STN **DUPLICATE 8**

2002415044 EMBASE ACCESSION NUMBER:

TITLE:

Neurite outgrowth promoting effect of cytokines on cultured

rat embryonic ventral spinal cord neurons.

AUTHOR:

Ichikawa Y.

7

CORPORATE SOURCE:

Y. Ichikawa, 4th Department of Internal Medicine, Toho

University School of Medicine, Chiba-ken, Japan

SOURCE:

Journal of the Medical Society of Toho University, (2002)

Vol. 49, No. 4-5, pp. 258-264. .

Refs: 25

ISSN: 0040-8670 CODEN: TOIZAG

COUNTRY:

Japan

DOCUMENT TYPE:

Journal; Article

FILE SEGMENT:

Neurology and Neurosurgery 800

Immunology, Serology and Transplantation 026

LANGUAGE:

Japanese

SUMMARY LANGUAGE:

English; Japanese

ENTRY DATE:

Entered STN: 2 Dec 2002

Last Updated on STN: 2 Dec 2002

Background: To clarify the possible neurite outgrowth promoting effect AB (NPE) of cytokines including interleukin-3 (IL-3), IL-6, erythropoietin (EPO), granulocyte-colony stimulating factor (G-CSF) and tumor necrosis factor- β (TNF- β) on cultured ventral spinal cord neurons of rat embryos, I studied the NPE effect of these cytokines on the primary explant cultures of ventral spinal cord neurons of fetal rats. Methods: The spinal cords from 13 or 14 embryonic day rat embryos were explanted. Explants in the culture medium were added cytokines at different concentrations, given on the first day of culture in the form of a single administration. For quantitative analysis of the NPE of these cytokines, neurite length was directly measured at the 7th culture day. Results: Neurite length was significantly increased in IL-3, IL-6 and EPO treated cultures and their effects seemed to be concentration-dependent in their effective concentration ranges. However, G-CSF and TNF- β had no neurite elongation effects at any concentration. Conclusion: These results suggest that several cytokines may have therapeutic potential in damaged motor neuron disorders, such as amyotrophic lateral sclerosis.

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reserved on STN

ACCESSION NUMBER: 2002323900 EMBASE

TITLE: Cytokines in coagulation and thrombosis: A preclinical and

clinical review.

AUTHOR: Joseph L.; Fink L.M.; Hauer-Jensen M.

CORPORATE SOURCE: Dr. L. Joseph, Department of Pathology, Univ. of Arkansas

for Med. Sciences, 4301 West Markham, Little Rock, AR

72205, United States. josephlija@uams.edu

SOURCE: Blood Coagulation and Fibrinolysis, (2002) Vol. 13, No. 2,

pp. 105-116. . Refs: 103

ISSN: 0957-5235 CODEN: BLFIE7

COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy

025 Hematology

026 Immunology, Serology and Transplantation

037 Drug Literature Index038 Adverse Reactions Titles

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 26 Sep 2002

Last Updated on STN: 26 Sep 2002

The cytokine network is a complex and dynamic system, involved in numerous AB biological responses in the human body. This review of the current literature describes the role of cytokines and their interaction with the coagulation system, specifically in the maintenance of the thrombo-hemorrhagic balance in vivo in human subjects and in animals. general, cytokines are thrombogenic, but they are amenable to therapeutic manipulations and hence are a potentially attractive tool in the clinician's armamentarium. Studies of the effects of cytokines in vivo are difficult because cytokines act in a very finite microenvironment and, although their actions are significant, they are transient. Most of the available clinical data related to interactions between cytokines and the coagulation system focuses on the role of tumor necrosis factor-alpha and interleukin-1 in septicemia and septic shock. However, several other cytokines and related proteins, such as platelet activating factor and plasminogen activator inhibitor, are also known to influence coagulation These factors interact closely with cytokines, and have and thrombosis. been included in this review for a better understanding of their interactions with traditional cytokines. Studies that utilize cell culture systems do not accurately model the in vivo status of this complex system and, hence, this review has excluded such studies. The role of the cytokine network in coronary artery disease, angiogenesis, or neoplasia has been addressed elsewhere by other workers and is not discussed here. By emphasizing important in vivo interactions, the intention of this review is to serve as an impetus to further translational research, both clinical and in the laboratory. .COPYRGT. 2002 Lippincott Williams & Wilkins.

L6 ANSWER 74 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER:

2002345430 EMBASE

TITLE:

[Embryonal stem cells].
EMBRYONALE STAMMZELLEN.

AUTHOR: Wurm T.

CORPORATE SOURCE:

Dr. T. Wurm, Senefelderstrasse 38, 94036 Passau, Germany Deutsche Apotheker Zeitung, (5 Sep 2002) Vol. 142, No. 36,

pp. 52-61. Refs: 11

ISSN: 0011-9857 CODEN: DAZEA2

COUNTRY:

SOURCE:

Germany

DOCUMENT TYPE: Journal; General Review FILE SEGMENT: 006 Internal Medicine

008 Neurology and Neurosurgery

021 Developmental Biology and Teratology

037 Drug Literature Index 049 Forensic Science Abstracts

LANGUAGE: German

ENTRY DATE: Entered STN: 17 Oct 2002

Last Updated on STN: 17 Oct 2002

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 75 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN

ACCESSION NUMBER: 2003:356961 BIOSIS DOCUMENT NUMBER: PREV200300356961

TITLE: Defective Yolk Sac Erythropoiesis in HIF-la-Null Mice: A

Role of Iron.

AUTHOR(S): Pastore, Yves D. [Reprint Author]; Divoky, Vladimir

[Reprint Author]; Liu, Enli [Reprint Author]; Ponka, Premysl [Reprint Author]; Semenza, Gregg L. [Reprint

Author]; Prchal, Josef T. [Reprint Author]

CORPORATE SOURCE: Medicine and Pediatric Hematology/Oncology, Baylor College

of Medicine, Houston, TX, USA

SOURCE: Blood, (November 16 2002) Vol. 100, No. 11, pp. Abstract

No. 2827. print.

Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002.

American Society of Hematology. CODEN: BLOOAW. ISSN: 0006-4971.

DOCUMENT TYPE: Conference; (Meeting)

Conference; (Meeting Poster)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 6 Aug 2003

Last Updated on STN: 6 Aug 2003

Hypoxia-inducible factor-1 (HIF-1) regulates the expression of an array of genes including erythropoietin (EPO), vascular endothelial growth factor (VEGF), transferrin and its receptor (TfR); however, its role in the erythropoiesis remains to be defined. In Hifla-/- mice, targeted disruption of the Hifla gene encoding the O2-regulated HIF-1a subunit causes cardiac malformations, vascular regression, and death by embryonic day (ED) 10.5. In order to understand the role of HIF-1a in erythropoiesis, we studied the yolk sac (YS) erythroid progenitors from Hifla-/-, Hifla+/-, and wild-type (WT) littermate embryos. Hematopoietic progenitors from isolated YS at ED 9-9.5 were analyzed by in vitro culture in presence of interleukin-3 (IL-3), IL-6, Epo, stem cell factor and granulocyte-macrophage colony stimulating factor

(GM-CSF). The numbers and the size of the erythroid colonies (CFU-E and BFU-E) from the YS of Hifla-/- embryos were decreased, and had a marked defect of hemoglobinization compared to erythroid colonies from WT and Hifla+/- YS. Neither VEGF nor high levels of Epo added to the cultures fully rescued these defects. Some of the differences might be related to a developmental arrest in the mutant embryos, as the number of somites in Hifla-/- embryos was significantly lower compared to the Hifla+/- and WT embryos (12, 20, 22 respectively). However, we hypothesized that the defective hemoglobinization may be due to abnormalities in iron metabolism, e.g. down-regulation of TfR. We cultured YS cells in the presence of salicylaldehyde isonicotinoyl hydrazone saturated with iron (Fe-SIH) which transports iron into cells independently of the TfR. In the presence of 100 micromolar Fe-SIH, we observed a significant increase in the numbers of erythroid colonies derived from YS of WT and, to a lesser degree, Hifla+/- embryos. Although there was no increase in the number of colonies from the YS of Hifla-/- embryos, the size of the

erythroid colonies and the degree of hemoglobinization was markedly improved. These results demonstrate defects in YS erythroid colony formation and hemoglobinization in Hifla-/- embryos and suggest that the latter may be associated with a disturbance of iron metabolism.

L6 ANSWER 76 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2001:338762 CAPLUS

DOCUMENT NUMBER:

134:362292

TITLE:

Methods of determining individual hypersensitivity to

a pharmaceutical agent from gene expression profile

INVENTOR(S): Farr, Spencer

PATENT ASSIGNEE(S): Phase-1 Molecular Toxicology, USA

CODEN: PIXXD2

SOURCE:

PCT Int. Appl., 222 pp.

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

F	PATENT NO.					KIN	D	DATE]	APPL:	ICAT	ION 1	NO.		D	ATE	
<u>-</u> W	10	2001	03292	28		A2	_	2001	0510	١	WO 2	000-1	US30	474		2	0001	103
M	O	2001	03292	2.8		A 3		2002	0725			•						
		W:	AE,	AG,	AL,	AM,	AT,	AU,	AZ,	BA,	BB,	BG,	BR,	BY,	BZ,	CA,	CH,	CN,
			CR,	CU,	CZ,	DE,	DK,	DM,	DZ,	EE,	ES,	FI,	GB,	GD,	GE,	GH,	GM,	HR,
			HU,	ID,	IL,	IN,	IS,	JP,	.KE,	KG,	KP,	KR,	KZ,	LC,	LK,	LR,	LS,	LT,
			LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NO,	NZ,	PL,	PT,	RO,	RU,
			SD,	SE,	SG,	SI,	SK,	SL,	ТJ,	TM,	TR,	TT,	TZ,	UA,	ŪĠ,	US,	UZ,	VN,
			YU,	ZA,	ZW,	AM,	AZ,	BY,	KG,	KZ,	MD,	RU,	TJ,	TM				
		RW:	GH,	GM,	KE,	LS,	MW,	MZ,	SD,	SL,	SZ,	TZ;	UG,	ZW,	AT,	BE,	CH,	CY,
			DE,	DK,	ES,	FI,	FR,	GB,	GR,	ΙE,	IT,	LU,	MC,	NL,	PT,	SE,	TR,	BF,
			ВJ,	CF,	CG,	CI,	CM,	GA,	GN,	GW,	ML,	MR,	NE,	SN,	TD,	TG		
PRIORI	PRIORITY APPLN. INFO.:				. :					1	US 1	999-	1653	98P		P 1	9991	105
										1	US 2	000-	1965	71P		P 2	0000	411

The invention discloses methods, gene databases, gene arrays, protein AB . arrays, and devices that may be used to determine the hypersensitivity of individuals to a given agent, such as drug or other chemical, in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes associated with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes associated with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes associated with hypersensitivity. The expression of the genes predetd, to be associated with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and apparatus useful for identifying hypersensitivity in a subject are also disclosed.

L6 ANSWER 77 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002351769 EMBASE

TITLE: Pharmacologically regulated cell therapy.

AUTHOR: Neff T.; Blau C.A.

CORPORATE SOURCE: C.A. Blau, Mailstop 357710, Health Sciences Building,

University of Washington, Seattle, WA 98195, United States.

tblau@u.washington.edu

SOURCE: Blood, (1 May 2001) Vol. 97, No. 9, pp. 2535-2540. .

Refs: 67

ISSN: 0006-4971 CODEN: BLOOAW

COUNTRY:

United States

DOCUMENT TYPE:

Journal: General Review Human Genetics 022

FILE SEGMENT:

025 Hematology

Immunology, Serology and Transplantation 026

Drug Literature Index 037 Adverse Reactions Titles 038

LANGUAGE:

English

ENTRY DATE:

Entered STN: 31 Oct 2002

Last Updated on STN: 31 Oct 2002

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

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reserved on STN

ACCESSION NUMBER: 2001018704 EMBASE

TITLE:

Paying for the greying of society.

AUTHOR:

Stokes R.

SOURCE:

Pharmaceutical Technology Europe, (2001) Vol. 13, No. 1,

pp. 58+60. .

ISSN: 0164-6826 CODEN: PTEUFB

COUNTRY:

United Kingdom

DOCUMENT TYPE:

Journal; (Short Survey)

FILE SEGMENT:

Gerontology and Geriatrics 020

Health Policy, Economics and Management 036

Cardiovascular Diseases and Cardiovascular Surgery 018

Urology and Nephrology 028

Endocrinology 003

037

Drug Literature Index

Neurology and Neurosurgery 800

LANGUAGE:

English English

SUMMARY LANGUAGE: ENTRY DATE:

Entered STN: 25 Jan 2001

Last Updated on STN: 25 Jan 2001

A recent US report has highlighted how all Western governments may need to AB work with insurers to make biotech-based drugs affordable for an ageing

population.

EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L6 ANSWER 79 OF 113 reserved on STN

ACCESSION NUMBER:

2000305414 EMBASE

TITLE:

The use of erythropoietin in neonates.

AUTHOR:

Ohls R.K.

CORPORATE SOURCE:

Dr. R.K. Ohls, Department of Pediatrics, ACC-3W, Univ. of New Mexico Hlth. Sci. Ctr., Albuquerque, NM 87131-5311,

United States. rohls@unm.edu

SOURCE:

Clinics in Perinatology, (2000) Vol. 27, No. 3, pp.

681-696. .

Refs: 80

ISSN: 0095-5108 CODEN: CLPEDL

COUNTRY:

United States

DOCUMENT TYPE:

Journal: General Review

FILE SEGMENT:

Pediatrics and Pediatric Surgery 007

Hematology 025

Health Policy, Economics and Management 036

Drug Literature Index 037 038 Adverse Reactions Titles

LANGUAGE:

English

SUMMARY LANGUAGE:

English

ENTRY DATE:

Entered STN: 14 Sep 2000

Last Updated on STN: 14 Sep 2000

Although much information has been accumulated about the clinical use of ABEpo in preterm infants, many questions remain unanswered. The evolution of clinical practice in the care of extremely ill, preterm infants

continues to affect the number of transfusions required during hospitalization. Decreasing phlebotomy losses and instituting standardized transfusion guidelines have both been shown significantly to decrease the transfusion requirements of preterm infants. The administration of Epo likely decreases transfusion need even further; however, the direct impact of each of these actions has not been studied prospectively. It is likely that the combination of instituting rigorous and standardized transfusion guidelines, decreasing phlebotomy losses, and the appropriate use of Epo will have the greatest impact in decreasing transfusion requirements in all preterm and term neonates, regardless of the cause of their anemia.

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                has been enhanced and reloaded
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NEWS 4
NEWS 5 NOV 03 JAPIO enhanced with IPC 8 features and functionality
NEWS 6 NOV 10 CA/CAplus F-Term thesaurus enhanced
        NOV 10
                STN Express with Discover! free maintenance release Version
NEWS
                8.01c now available
        NOV 20 CAS Registry Number crossover limit increased to 300,000 in
NEWS 8
                additional databases
        NOV 20 CA/CAplus to MARPAT accession number crossover limit increased
NEWS
                to 50,000
NEWS 10 DEC 01 CAS REGISTRY updated with new ambiguity codes
NEWS 11 DEC 11 CAS REGISTRY chemical nomenclature enhanced
NEWS 12 DEC 14 WPIDS/WPINDEX/WPIX manual codes updated
        DEC 14
                GBFULL and FRFULL enhanced with IPC 8 features and
NEWS 13
                functionality
                CA/CAplus pre-1967 chemical substance index entries enhanced
NEWS 14
        DEC 18
                with preparation role
                CA/CAplus patent kind codes updated
        DEC 18
NEWS 15
NEWS 16
                MARPAT to CA/CAplus accession number crossover limit increased
        DEC 18
                to 50,000
                MEDLINE updated in preparation for 2007 reload
        DEC 18
NEWS 17
                CA/CAplus enhanced with more pre-1907 records
NEWS 18
        DEC 27
                CHEMLIST enhanced with New Zealand Inventory of Chemicals
         JAN 08
NEWS 19
NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT
             MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
             AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.
NEWS HOURS
              STN Operating Hours Plus Help Desk Availability
             Welcome Banner and News Items
NEWS LOGIN
             For general information regarding STN implementation of IPC 8
NEWS IPC8
             X.25 communication option no longer available
NEWS X25
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FILE 'HOME' ENTERED AT 10:58:32 ON 11 JAN 2007

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=> file medline embase biosis caplus
COST IN U.S. DOLLARS
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TOTAL SINCE FILE ENTRY SESSION 0.21 0.21

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 10:58:48 ON 11 JAN 2007

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FILE 'BIOSIS' ENTERED AT 10:58:48 ON 11 JAN 2007 Copyright (c) 2007 The Thomson Corporation

FILE 'CAPLUS' ENTERED AT 10:58:48 ON 11 JAN 2007 USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT. PLEASE SEE "HELP USAGETERMS" FOR DETAILS. COPYRIGHT (C) 2007 AMERICAN CHEMICAL SOCIETY (ACS)

=> s (GMCSF or granulocyte(w) macrophage(w) colony(w) stimulating(w) factor or GCSF granulocyte(w) colony(w) stimulating(w) factor)

69446 (GMCSF OR GRANULOCYTE(W) MACROPHAGE(W) COLONY(W) STIMULATING(W) Ll FACTOR OR GCSF GRANULOCYTE(W) COLONY(W) STIMULATING(W) FACTOR)

=> s ll and (ischemia or hypoxia or stroke or neurodegenerative or neurological) 672 L1 AND (ISCHEMIA OR HYPOXIA OR STROKE OR NEURODEGENERATIVE OR L2NEUROLOGICAL)

=> s 12 and treatment

240 L2 AND TREATMENT L3

=> s 13 and (interleuikin or erythropoietin)

23 L3 AND (INTERLEUIKIN OR ERYTHROPOIETIN) L4

=> dup rem 14

PROCESSING COMPLETED FOR L4

23 DUP REM L4 (0 DUPLICATES REMOVED) L5

=> dis his

(FILE 'HOME' ENTERED AT 10:58:32 ON 11 JAN 2007)

FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 10:58:48 ON 11 JAN 2007 69446 S (GMCSF OR GRANULOCYTE(W) MACROPHAGE(W) COLONY(W) STIMULATING(W) F L1672 S L1 AND (ISCHEMIA OR HYPOXIA OR STROKE OR NEURODEGENERATIVE OR L2 240 S L2 AND TREATMENT L3

23 S L3 AND (INTERLEUIKIN OR ERYTHROPOIETIN) L423 DUP REM L4 (0 DUPLICATES REMOVED) L5

=> dis ibib abs 15 15-23

CAPLUS COPYRIGHT 2007 ACS on STN ANSWER 15 OF 23 L5

ACCESSION NUMBER:

2003:282718 CAPLUS

DOCUMENT NUMBER:

138:282352

TITLE:

Traversal of nucleic acid molecules through a tissue

fluid space and expression in repair cells

INVENTOR(S):

Sosnowski, Barbara A.; Pierce, Glenn

PATENT ASSIGNEE(S):

Selective Genetics, Inc., USA PCT Int. Appl., 95 pp.

SOURCE:

CODEN: PIXXD2

DOCUMENT TYPE:

Patent English

LANGUAGE: FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

```
APPLICATION NO.
                                                                   DATE
     PATENT NO.
                         KIND
                                DATE
                                            WO 2002-US31546
                                                                    20021002
    WO 2003029429
                          A2
                                20030410
                          A3
                                20040401
    WO 2003029429
                          A9
                                20040701
    WO 2003029429
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
        RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF,
             CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
                                           AU 2002-343475
                                                                    20021002
     AU 2002343475
                                20030414
                          A1
                                                                    20021002
                                20030807
                                            US 2002-264284
                          A1
     US 2003148979
                                            EP 2002-780419
                                                                    20021002
                          A2
                                20040721
     EP 1438413
            AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
         R:
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK
                                                                    20011003
                                            US 2001-327513P
                                                                 P
PRIORITY APPLN. INFO.:
                                            WO 2002-US31546
                                                                    20021002
```

AB Disclosed are methods for use in transferring nucleic acids into cells at a wound site associated with a fluid space. These gene transfer protocols are suitable for use in transferring various nucleic acids into cartilage, cardiac muscle, and other tissues, and have many uses including treating diseases such as arthritis and ischemic heart disease, and promoting wound healing. The invention further disclosed pharmaceutical compns. that may be used in the practice of the invention to transfer the nucleic acid of interest. Such compns. include any multi-partitioned biocompatible matrix in combination with multiple nucleic acids of interest. Thus, collagen collagen-immobilized fibroblast growth factor (FGF) genes induce angiogenesis in vitro, and FGF gene delivery to skeletal muscle wounds induces both angiogenesis and arteriogenesis and well as induces myocyte regeneration.

L5 ANSWER 16 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:678274 CAPLUS

DOCUMENT NUMBER: 139:193995

TITLE: Pluripotent embryonic-like stem cells from adult

tissues and their culture and therapeutic uses

INVENTOR(S): Young, Henry E.; Lucas, Paul A.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 186 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003161817	A1	20030828	US 2001-820320	20010328
US 2005255588	A1	20051117	US 2005-29763	20050105
PRIORITY APPLN. INFO.:			US 2001-820320 B1	20010328
	77 24	- la	-imiles to pluripotont	ombrachic

AB Pluripotent stem cells with properties similar to pluripotent embryonic stem cells are purified from adult tissues for therapeutic and investigative use. The invention further relates to methods of purifying pluripotent embryonic-like stem cells and to compns., cultures and clones thereof. The present invention also relates to a method of transplanting the pluripotent stem cells of the present invention in a mammalian host, such as human, comprising introducing the stem cells, into the host. The invention further relates to methods of in vivo administration of a

protein or gene of interest comprising transfecting a pluripotent stem cell with a construct comprising DNA which encodes a protein of interest and then introducing the stem cell into the host where the protein or gene of interest is expressed. The present also relates to methods of producing mesodermal, endodermal or ectodermal lineage-committed cells by culturing or transplantation of the pluripotent stem cells of the present invention.

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CAPLUS COPYRIGHT 2007 ACS on STN
    ANSWER 17 OF 23
L5
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2003:1013125 CAPLUS ACCESSION NUMBER:

140:65078 DOCUMENT NUMBER:

Reduced side-effect hemoglobin compositions TITLE:

Looker, Douglas L.; Apostol, Izydor Z.; Brucker, Eric INVENTOR(S): A.; Doyle, Michael P.; Foster, David L.; Glascock, Christopher B.; Hartman, James C.; Lee, Geoffrey F.; Lemon, Douglas D.; Moore, Edwin G.; Richards, Jane P.;

Schick, Michael R.; Trimble, Stephen P.; Pereira,

David; Hai, Ton-That; Burhop, Kenneth E.

Baxter International Inc., USA; Baxter Healthcare S.A. PATENT ASSIGNEE(S):

U.S., 62 pp., Cont.-in-part of U.S. 6,455,676. SOURCE:

CODEN: USXXAM

Patent DOCUMENT TYPE: English LANGUAGE:

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PAT	CENT	NO.			KIND DATE					APPLICATION NO.						DATE			
WO	6670 9850	B1 20031230 A2 19981112 A3 19990401			US 2000-709914 WO 1998-US8861						20001110 19980501								
WO		AL, DK, KP, NO, UA, GH,	EE, KR, NZ, UG, GM,	AT, ES, KZ, PL, US, KE,	AU, FI, LC, PT, UZ, LS,	AZ, GB, LK, RO, VN, MW,	BA, GE, LR, RU, YU, SD,	BB, GH, LS, SD, ZW SZ,	GM, LT, SE, UG,	GW , LU , SG ,	, BY, , HU, , LV, , SI,	ID, MD, SK, BE,	IL, MG, SL,	IS, MK, TJ,	JP, MN, TM,	KE, MW, TR,	KG, MX, TT,		
	6455 2004 7 APP	CM, 676 2597	GA,	GN,	ML, Bl	MR,	NE, 2002	SN, 0924	TD,	TG US 2 US 2	2000-4 2003-7 1998-1	4032 7475	08 80		2	00004 0031: 9980!	125 229		
				1					1	US 3 US 3	1999-1 2000-4 1997-4 1997-5	4032 4536 5798	08 4P 6P	1	A2 2 P 1 P 1	9991: 00004 9970: 9970: 0001:	125 502 905		

The invention relates to novel Hb compns., particularly novel recombinant AB mutant Hb compns., which eliminate or substantially reduce 1) the creation of heart lesions, 2) gastrointestinal discomfort, 3) pressor effects, and 4) endotoxin hypersensitivity associated with the administration of extracellular Hb compns. in various therapeutic applications. Applications described include treatments for anemia, head injury, hemorrhage or hypovolemia, ischemia, cachexia, sickle cell crisis and stroke; enhancing cancer treatments; stimulating hematopoiesis; improving repair of phys. damaged tissues; alleviating cardiogenic shock; and shock resuscitation. REFERENCE COUNT: THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS 41

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5ANSWER 18 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:573223 CAPLUS DOCUMENT NUMBER: 139:111699

Uses of G-CSF, GM-CSF and SCF in conjunction with TITLE:

other growth factors for the mobilization of stem cells as a new therapeutic approach to cerebrovascular

and spinal cord injury therapy Pourquier, Didier; Moukoko, Didier

INVENTOR(S):

PATENT ASSIGNEE(S): Fr.

SOURCE:

Fr. Demande, 24 pp.

CODEN: FRXXBL

DOCUMENT TYPE:

Patent French

LANGUAGE:

1

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

```
PATENT NO.
                         KIND
                                DATE
                                            APPLICATION NO.
                                                                    DATE
                         _ _ _ _
                                            FR 2002-610
                                                                    20020118
                          Al
                                20030725
    FR 2834898
    FR 2834898
                          B1.
                                20050610
                                            WO 2003-FR13
                          A1
                                20030731
                                                                    20030106
    WO 2003061685
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
        RW: GH, GM, KE, LS, MW, MZ, SD, SL; SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
                          A1
                                20041013
                                            EP 2003-712209
                                                                    20030106
     EP 1465653
            AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
                                             FR 2002-610
                                                                 A 20020118
PRIORITY APPLN. INFO.:
                                             WO 2003-FR13
                                                                    20030106
```

The invention relates to a new therapeutic application of at least of the factors chosen among G-CSF (granulocyte colony-stimulating factor), GM-CSF (macrophages-granulocytes colony-stimulating factor) and the SCF (stem cell factor). This factor is to be used in the preparation of a useful drug for auxiliary treatment leading to the reconstruction of nerve fibers. G-CSF, GM-CSF and SCF are particularly useful as drugs for the treatment of ischemic or hemorrhagic cerebrovascular accidents, cerebral traumas, ischemic or hemorrhagic vascular accidents of the spinal cord, and spinal cord traumas. Administration is intended in a general way, both for human and veterinary medicine.

REFERENCE COUNT:

THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER:

2001:360039 CAPLUS

DOCUMENT NUMBER:

134:371751

TITLE:

Reduced side-effect hemoglobin compositions

INVENTOR(S):

Looker, Douglas L.; Apostol, Izydor Z.; Brucker, Eric A.; Doyle, Michael P.; Foster, David L.; Glascock, Christopher B.; Hartman, James C.; Lee, Geoffrey F.; Lemon, Douglas D.; Moore, Edwin G.; Richards, Jane P.;

Schick, Michael R.; Trimble, Stephen P.; Pereira,

David; Hai, Ton-That; Burhop, Kenneth E.

PATENT ASSIGNEE(S):

Baxter Biotech Technology S.A.R.L., Switz.

SOURCE:

PCT Int. Appl., 146 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent English

LANGUAGE:

T: 3

FAMILY ACC. NUM. COUNT: PATENT INFORMATION:

PATENT NO.

KIND DATE

APPLICATION NO.

DATE

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20010517
                                            WO 2000-US30857
     WO 2001034648
                          Al
            AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
            CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
            HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
            LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
             SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
             YU, ZA, ZW
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             BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
                                20010517
                                            CA 2000-2391226
                          A1
                                                                    20001110
     CA 2391226
                          A1
                                20020828
                                            EP 2000-980318
                                                                    20001110
     EP 1233986 .
         R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
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                                                                    20001110
     JP 2003515533
                          T
                                20030507
                                            JP 2001-537359
     AU 784195
                          B2
                                20060216
                                            AU 2001-17597
                                                                    20001110
                                20020711
                                           NO 2002-2229
                                                                    20020510
     NO 2002002229
                       . A
                                                                    20020514
                                20030228
                                            ZA 2002-3817
     ZA 2002003817
                          Α
                                            US 1999-165289P
                                                                P 19991112
PRIORITY APPLN. INFO.:
                                            WO 2000-US30857
                                                                    20001110 .
                                                                W
    The invention relates to novel Hb compns., particularly novel recombinant
     mutant Hb compns., which eliminate or substantially reduce 1) the creation
     of heart lesions, 2) gastrointestinal discomfort, 3) pressor effects, and
     4) endotoxin hypersensitivity associated with the administration of
     extracellular Hb compns. in various therapeutic applications.
     Applications described include treatments for anemia, head
     injury, hemorrhage or hypovolemia, ischemia, cachexia, sickle
     cell crisis and stroke; enhancing cancer treatments;
     stimulating hematopoiesis; improving repair of phys. damaged tissues;
     alleviating cardiogenic shock; and shock resuscitation.
                               THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS
REFERENCE COUNT:
                         1
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
     ANSWER 20 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights
L5
     reserved on STN
                    2000337455 EMBASE
ACCESSION NUMBER:
                    Treatment of leukemia, lymphoma and cancer - 13th
TITLE:
                    International Symposium: Molecular Biology of
                    Hematopoiesis: 14-18 July 2000, New York, NY, USA.
                    Rose-John S.
AUTHOR:
                    S. Rose-John, Department of Biochemistry,
CORPORATE SOURCE:
                    Christian-Albrechts Universitat Kiel, Olshausenstrasse 40,
                    D-24098 Kiel, Germany. rosejohn@biochem.uni-kiel.de
                    Current Opinion in Oncologic, Endocrine and Metabolic
SOURCE:
                    Investigational Drugs, (2000) Vol. 2, No. 4, pp. 423-425. .
                    ISSN: 1464-8466 CODEN: COODF2
                    United Kingdom
COUNTRY:
                    Journal; Conference Article
DOCUMENT TYPE:
                            Drug Literature Index
                    037
FILE SEGMENT:
                    016
                            Cancer
                    025
                            Hematology
                    022
                            Human Genetics
                            Pharmacology
                    030
                            Immunology, Serology and Transplantation
                    026
                    English
LANGUAGE:
                    English
SUMMARY LANGUAGE:
                    Entered STN: 13 Oct 2000
ENTRY DATE:
                    Last Updated on STN: 13 Oct 2000
     This conference included sessions covering the clinical aspects of
AB
     methodological CD34+ cell expansion, identification of the true
     hematopoietic stem cells, cancer and coagulation therapies, pathology of
     anemia in cancer patients following chemotherapy, molecular biology of
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erythropoietin signaling, and the role of hypoxia as a

regulator of malignant cell growth. Sessions on lymphopoiesis, dendritic cells and the control of the immune response were presented in addition to updates on the JAK/STAT signaling pathways of cytokine receptors and on the construction and use of novel designer cytokines in the expansion of hematopoietic progenitor cells and gene therapy of human cancer. Innovation and new strategies for bone marrow transplantation and organ transplants were discussed, and the use of p53 gene transfer in the induction of apoptosis in malignant cells was evaluated. More than 400 scientific contributions were presented as plenary talks, short communications and poster presentations; approximately 250 to 300 people attended.

L5 ANSWER 21 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000081357 EMBASE

TITLE: Manifestations and treatment of Schimke

immuno-osseous dysplasia: 14 new cases and a review of the

literature.

AUTHOR: Boerkoel C.F.; O'Neill S.; Andre J.L.; Benke P.J.;

Bogdanovic R.; Bulla M.; Burguet A.; Cockfield S.; Cordeiro I.; Ehrich J.H.H.; Frund S.; Geary D.F.; Ieshima A.; Illies F.; Joseph M.W.; Kaitila I.; Lama G.; Leheup B.; Ludman M.D.; McLeod D.R.; Medeira A.; Milford D.V.; Ormala T.; Rener-Primec Z.; Santava A.; Santos H.G.; Schmidt B.; Smith

G.C.; Spranger J.; Zupancic N.; Weksberg R.

CORPORATE SOURCE: R. Weksberg, Hospital for Sick Children, Div. of

Clinic./Metabolic Genetics, University of Toronto, 555

University Avenue, Toronto, Ont. M5G 1X8, Canada.

rweksbq@sickkids.on.ca

SOURCE: European Journal of Pediatrics, (2000) Vol. 159, No. 1-2,

pp. 1-7. . Refs: 23

ISSN: 0340-6199 CODEN: EJPEDT

COUNTRY: Germany

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 007 Pediatrics and Pediatric Surgery

022 Human Genetics

037 Drug Literature Index

LANGUAGE: English
SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 16 Mar 2000

Last Updated on STN: 16 Mar 2000

AB Schimke immuno-osseous dysplasia (SIOD) is a rare autosomal recessive spondylo-epiphyseal dysplasia. The characteristic features of SIOD include 1) short stature with hyperpigmented macules and an unusual facies, 2) proteinuria with progressive renal failure, 3) lymphopenia with recurrent infections, and 4) cerebral ischaemia. Although 25 patients have been reported with this disorder, the clinical course and phenotype of SIOD are not well characterized. This report summarizes the clinical findings, course and treatment of reported patients and includes 14 additional patients with SIOD. We emphasize the high incidence of cerebral ischaemia and ocular abnormalities, define the high incidence of thyroid dysfunction and blood cytopenia, and confirm the absence of effective and durable medical therapies. Conclusion: Schimke immuno-osseous dysplasia is a multi-system autosomal recessive disorder with variable expression that affects the skeletal, renal, immune, vascular, and haematopoietic systems. Medical therapy is limited especially for more severely affected individuals.

L5 ANSWER 22 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1998:323165 CAPLUS

DOCUMENT NUMBER: 129:8577

TITLE: Methods for regulating angiogenesis INVENTOR(S): Isner, Jeffrey M.; Asahara, Takayuki

PATENT ASSIGNEE(S): St. Elizabeth's Medical Center of Boston, Inc., USA

SOURCE: PCT Int. Appl., 57 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

	PA	TENT	NO.			KINI)	DATE		API	PLICAT	'ION NO.		Ι	DATE	•	
	WO	9819	712			A1	-	1998	0514	WO	1997-	US19935		-	9971	106	
			AU,	•													
		RW:	AT,	BE,	CH,	DE,	DK	, ES,	ĖΙ,	FR, GI	3, GR,	IĒ, IT,	LU,	MC,	NL,	PT,	SE
	US	5980	887			Α		1999	1109	US	1996-	744882		-	19961	108	
	CA	2271	690			A1		1998	0514	CA	1997-	2271690		-	19971	106	
	AU	9852	432			A		1998	0529	AU	1998-	52432		-	L9971	106	
	AU	7432	67			B2		2002	0124								
	EP	9411	25			Al		1999	0915	EP	1997-	947319			19971	106	
		R:	DE,	FR,	GB,	IT											
	JP	2001	5034	27		${f T}$		2001	0313	JP	1998-	521645			L9971	106	
	EP	1618	898			A2		2006	0125	EP	2005-	17496			19971	106	
		R:	DE,	FR,	GB,	IT											
PRIO	RIT	Y APP	•	•	•					US	1996-	744882	Į	<i>.</i>	19961	108	
7										EP	1997-	947319	7	43 :	19971	106	
										WO	1997-	US19935	V	v :	L9971	106	

AB In accordance with the present invention, EC (endothelial cell) progenitors can be used in a method for regulating angiogenesis, i.e., enhancing or inhibiting blood vessel formation, in a selected patient and in some preferred embodiments for targetting specific locations. For example, the EC progenitors can be used to enhance angiogenesis or to deliver an angiogenesis modulator, e.g. anti- or pro-angiogenic agents, resp. to sites of pathol. or utilitarian angiogenesis. Addnl., in another embodiment, EC progenitors can be used to induce reendothelialization of an injured blood vessel, and thus reduce restenosis by indirectly inhibiting smooth muscle cell proliferation.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 23 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1998359165 EMBASE

TITLE: Strategies for the use of epoetin alfa in breast cancer

patients.

AUTHOR: Del Mastro L.; Venturini M.

CORPORATE SOURCE: Dr. L. Del Mastro, Oncologia Medica 1, Isto. Nazionale

Ricerca Cancro, L. go Rosanna Benzi 10, 16132 Genova,

Italy. mventur@hp380.ist.unige.it

SOURCE: Oncologist, (1998) Vol. 3, No. 5, pp. 314-318.

Refs: 27

ISSN: 1083-7159 CODEN: OCOLF6

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer

025 Hematology

030 Pharmacology

037 Drug Literature Index 038 Adverse Reactions Titles

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 19 Nov 1998

Last Updated on STN: 19 Nov 1998

AB Anemia is a common complication in cancer patients undergoing chemotherapy, and its severity depends on both the type of antineoplastic drugs and the clinical status of the patient. Breast cancer patients

undergoing standard chemotherapy develop clinically significant anemia in up to 25% of cases. This percentage, moreover, increases up to 63% when more intensive chemotherapy regimens are used. The therapeutic use of erythropoietin in anemic patients, i.e., in patients with hemoglobin levels below 9-10.5 g/dl, is able to correct the anemic status in nearly 40%-80% of such patients, but it does not completely eliminate the need of blood transfusions: 20%-40% of patients need to be transfused despite the erythropoietin treatment. An alternative strategy for optimizing the erythropoietin treatment is its use in the prevention of anemia, i.e., in patients with normal hemoglobin values but at high risk of becoming anemic. In a phase III study, we evaluated the role of erythropoietin in the prevention of anemia in breast cancer patients undergoing dose-intensive chemotherapy. Clinically significant anemia occurred in 52% (95% CI = 33-69) of control patients and in no patient (95% CI = 0-14) in the erythopoietin arm (p = .00001). After six cycles of chemotherapy the mean hemoglobin decrease was 3.05 g/dl (\pm 1.0, 95% CI = 2.6-3.5) in the control arm and 0.8 g/dl (\pm 1.4, 95% CI = 0.3-1.4) in the erythropoietin arm. Moreover, 6.4% of control patients needed blood transfusion compared to no patients in the erythropoietin Erythropoietin is active in both the treatment and the prevention of anemia in cancer patients undergoing chemotherapy. Due to its high economic cost, efforts should be made to identify subsets of patients in whom the preventive use could be cost-effective. Patients undergoing chemotherapy associated with a high risk of anemia could benefit from preventive use of erythropoietin in special circumstances, such as presence of risk of myocardial or cerebral ischemia, uncommon blood group, or religious beliefs hindering blood transfusions. Moreover, anemia prevention could be considered in patients at high risk of requiring blood transfusions, such as patients with low baseline value of hemoglobin or with a hemoglobin decrease of ≥2 g/dl after the first cycle of chemotherapy.

=> dis ibib abs 15 1-14

L5 ANSWER 1 OF 23 MEDLINE on STN ACCESSION NUMBER: 2006429365 MEDLINE DOCUMENT NUMBER: PubMed ID: 16856086

TITLE: Colony stimulating factors (including

erythropoietin, granulocyte colony stimulating

factor and analogues) for stroke.

AUTHOR: Bath P M W; Sprigg N

CORPORATE SOURCE: University of Nottingham, Division of Stroke Medicine,

South Block D Floor, Queens Medical Centre, Nottingham, Nottinghamshire, UK NG7 2UH. philip.bath@nottingham.ac.uk

SOURCE: Cochrane database of systematic reviews (Online), (2006)

Vol. 3, pp. CD005207. Electronic Publication: 2006-07-19.

Ref: 27

Journal code: 100909747. E-ISSN: 1469-493X.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200610

ENTRY DATE: Entered STN: 21 Jul 2006

Last Updated on STN: 17 Oct 2006 Entered Medline: 16 Oct 2006

AB BACKGROUND: Colony stimulating factors (CSFs), also called haematopoietic growth factors, regulate bone marrow production of circulating red and white cells, and platelets. They have been shown to be neuroprotective in experimental stroke. Some CSFs also mobilise the release of bone marrow stem cells into the circulation. OBJECTIVES: We

systematically assessed the effects of CSFs on functional outcome and haematology measures in patients with acute or subacute stroke enrolled into randomised controlled trials. SEARCH STRATEGY: We searched the Cochrane Stroke Group Trials Register (last searched February 2005), the Cochrane Central Register of Controlled Trials (CENTRAL) (The Cochrane Library Issue 3, 2005), MEDLINE (1985 to March 2006), EMBASE (1985 to November 2005), and Science Citation Index (1985 to November 2005). In an attempt to identify further published, unpublished and ongoing trials we contacted manufacturers and principal investigators of trials (last contacted 2005). We also searched reference lists of relevant articles and reviews. SELECTION CRITERIA: Unconfounded randomised controlled trials recruiting patients with acute or subacute ischaemic or haemorrhagic stroke were included. CSFs included stem cell factor (SCF), erythropoietin (EPO), granulocyte colony stimulating factor (G-CSF), granulocyte-macrophage colony stimulating factor (GM-CSF), macrophage-colony stimulating factor (M-CSF, CSF-1), and thrombopoietin (TPO), or analogues of these. The primary outcome was functional outcome (assessed as combined death or disability and dependency using scales such as the modified Rankin Scale or Barthel Index) at the end of the trial. Secondary outcomes included safety at the end of treatment (death, impairment, deterioration, extension or recurrence), death at the end of follow up, and haematology measures (blood counts at or around day seven after treatment commenced). DATA COLLECTION AND ANALYSIS: Data on measures by intention to treat (where available) were collected and analysed as dichotomous or continuous outcomes, as relevant, using random-effects models. Heterogeneity was assessed. MAIN RESULTS: No large trials were identified. EPO therapy was associated with a non-significant reduction in neurological impairment in one small trial (n = 40 participants) but had no significant effect on haematological measures. Further small trials of EPO and G-CSF are ongoing. AUTHORS' CONCLUSIONS: No large trials of EPO, G-CSF or other colony stimulating factors have been performed and it is too early to know whether CSFs improve functional outcome.

L5 ANSWER 2 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006323695 EMBASE

TITLE: Hematopoietic colony stimulating factors in cardiovascular

and pulmonary remodeling: Promoters or inhibitors?.

AUTHOR: Parissis J.; Filippatos G.; Adamopoulos S.; Li X.;

Kremastinos D.Th.; Uhal B.D.

CORPORATE SOURCE: B.D. Uhal, Department of Physiology, Michigan State

University, 3185 Biomedical/Physical Sci. Bldg., East Lansing, MI 48824-3320, United States. uhal@msu.edu

SOURCE: Current Pharmaceutical Design, (2006) Vol. 12, No. 21, pp.

2689-2699. . Refs: 120

ISSN: 1381-6128 CODEN: CPDEFP

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis

018 Cardiovascular Diseases and Cardiovascular Surgery

1025 Hematology
030 Pharmacology

037 Drug Literature Index 038 Adverse Reactions Titles

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 27 Jul 2006

Last Updated on STN: 27 Jul 2006

AB Hemopoietic colony stimulating factors (HCSFs) are naturally occurred substances that are released in response to infection or inflammation and regulate the proliferation and differentiation of hemopoietic progenitor

Some representative members of this peptide family induce atherogenesis through the mediation of monocyte-endothelial cell adhesive interaction and promotion of angiogenesis within the atherosclerotic plaques. HCSFs, such as granulocyte-macrophage colony-stimulating factor (GM-CSF), also promote post-infarction cardiac remodeling though the enhanced activation and infiltration of monocytes into injured myocardial tissue and through altered equilibrium of collagen deposition/degradation. On the other hand, exogenous administration of granulocyte colony-stimulating factor (G-CSF) or eythropoietin (EPO) in patients with chronic ischemic disease or recent myocardial infarction have lead to beneficial arteriogenesis or myocardial cell regeneration, thus preventing adverse cardiac remodeling. While GM-CSF may hold therapeutic potential as an inhibitor of lung fibrogenesis, G-CSF appears to promote fibrosis in the lungs. pathophysiological role of HCSFs also depends on the timing of their action on cardiovascular remodeling, as well as on the target progenitor hematopoietic cell. This article summarizes current knowledge about the clinical and therapeutic implications of these factors in chronic artery disease, post-infarction cardiac remodeling, chronic heart failure and in pulmonary fibrosis. . COPYRGT. 2006 Bentham Science Publishers Ltd.

L5 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:1314101 CAPLUS

DOCUMENT NUMBER: 144:68263

TITLE: Genes showing altered levels of expression in

drug-resistant leukemia and their use in diagnosis and

selection of drug target for therapy

INVENTOR(S): Evans, William E.; Pieters, Rob; Cheok, Meyling H.;

Den Boer, Monique L.; Yang, Wenjian

PATENT ASSIGNEE(S): St. Jude Children's Research Hospital, USA; Erasmus

University Medical Center Rotterdam

SOURCE: PCT Int. Appl., 124 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PAT	ENT	NO.			KIND DATE				i	APPL:	ICAT:		DATE				
	-				·	-					~			-			
WO	2005118865				A2		2005	1215	Ţ	WO 2	005-1	JS1 _. 74	•	20050518			
WO	2005118865				A3		20060622						•				
	W:	AE,	AG,	AL,	AM,	AT,	AU,	AZ,	BA,	BB,	BG,	BR,	BW,	BY,	BZ,	CA,	CH,
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		LC,	LK,	LR,	LS,	LT,	LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NA,
		NG,	NI,	NO,	NZ,	OM,	PG,	PH,	PL,	PT,	RO,	RU,	SC,	SD,	SE,	SG,	SK,
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	RW:	ВW,	GH,	GM,	KE,	LS,	MW,	MZ,	NA,	SD,	SL,	SZ,	TZ,	UG,	ZM,	ZW,	AM,
		AZ,	BY,	KG,	KZ,	MD,	RU,	TJ,	TM,	AT,	BE,	BG,	CH,	CY,	CZ,	DE,	DK,
		EE,	ES,	FI,	FR,	GB,	GR,	HU,	ΙE,	IS,	IT,	LT,	LU,	MC,	NL,	PL,	PT,
		RO,	SE,	SI,	SK,	TR,	BF,	ВJ,	CF,	CG,	CI,	CM,	GA,	GN,	GQ,	GW,	ML,
		MR,	NE,	SN,	TD,	TG											
PRIORITY	APP	LN.	INFO	. : .					1	US 2	004-	5757	62P		P 20	0040	528

The present invention encompasses methods and compns. useful in the diagnosis and treatment of drug resistant leukemia. The invention provides a number of genes that are differentially expressed between drug resistant and drug sensitive acute lymphoblastic leukemia (ALL). These genes act as biomarkers for drug resistant leukemia, and further serve as mol. targets for drugs useful in treating drug resistant leukemia. Accordingly, the invention provides methods of diagnosing drug resistant leukemia and methods of selecting a therapy for subjects affected by drug-resistant leukemia. The invention also provides methods

for screening for compds. for treating drug-resistant leukemia, and improved methods for treating drug-resistant leukemia. Compns. of the invention include arrays, computer readable media, and kits for use in the methods of the invention

methods of the invention. CAPLUS COPYRIGHT 2007 ACS on STN ANSWER 4 OF 23 L5 2005:572333 CAPLUS ACCESSION NUMBER: DOCUMENT NUMBER: 143:91472 TITLE: Methods of treating neurological conditions with hematopoietic growth factors Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger, INVENTOR (S): Carola; Sommer, Clemens; Schwab, Stefan; Kollmar, Rainer; Maurer, Martin; Weber, Daniela; Gassler, Nikolaus PATENT ASSIGNEE(S): Axaron Bioscience Ag, Germany U.S. Pat. Appl. Publ., 169 pp., Cont.-in-part of Appl. SOURCE: No. PCT/IB03/06446. CODEN: USXXCO DOCUMENT TYPE: Patent English LANGUAGE: FAMILY ACC. NUM. COUNT: 2 PATENT INFORMATION: KIND APPLICATION NO. DATE PATENT NO. DATE Al 20050630 US 2004-880101 20040630 US 2005142102 US 2004141946 Al 20040722 US 2003-659295 20030911 WO 2004058287 A2 20040715 WO 2003-IB6446 20031231 WO 2004058287 8A 20041021 WO 2004058287 **A**3 20041216 AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG WO 2004-IB4329 20041229 WO 2006008582 **A1** 20060126 W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, OG, ZM, ZW, AM, AZ, B1, RG KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.:

US 2002-331755

B1 20021231

US 2003-659295

A2 20030911

WO 2003-IB6446 A2 20031231 US 2004-880101 A 20040630

The present invention relates to a method of treating a neurol. condition in a mammal by administering at least one hematopoietic growth factor from the group consisting of GCSF, GMCSF, IL-3, IL-5, a derivative thereof, or a mimetic thereof. A method is also claimed of treating a neurol. condition using neural stem cells treated with a hematopoietic factor. Also claimed is a method of enhancing the survival of a cell transplanted into a mammal, comprising introducing into the cell one or more polynucleotides which encode a hematopoietic factor.

A method of enhancing the viability of a neural cell culture comprising contacting the neural cell culture with a hematopoietic factor is addnl. claimed.

ANSWER 5 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L5 reserved on STN

2005304965 EMBASE ACCESSION NUMBER:

Thrombocytosis during antifungal therapy of candidemia. TITLE: Saathoff A.D.; Elkins S.L.; Chapman S.W.; McAllister S.F.; **AUTHOR:**

Cleary J.D.

Dr. J.D. Cleary, University of Mississippi Medical Center, CORPORATE SOURCE:

2500 N. State St., Jackson, MS 39216-4500, United States.

Jcleary@umsmed.edu

Annals of Pharmacotherapy, (2005) Vol. 39, No. 7-8, pp. SOURCE:

> 1238-1243. . Refs: 24

ISSN: 1060-0280 CODEN: APHRER

United States COUNTRY: DOCUMENT TYPE: Journal; Article Microbiology FILE SEGMENT: 004

> Internal Medicine 006

Hematology 025

Drug Literature Index 037 Adverse Reactions Titles 038

LANGUAGE: English

English; Spanish; French SUMMARY LANGUAGE: Entered STN: 29 Sep 2005 ENTRY DATE:

Last Updated on STN: 29 Sep 2005

BACKGROUND: Secondary, "reactive," thrombocytosis has been attributed to AB bacterial infection and treatment with multiple pharmaceuticals and may be associated with an increase in the incidence of gastrointestinal tract bleeding and thrombotic events (eg, stroke). OBJECTIVE: To characterize the dynamics of thrombocytosis in patients with candidemia receiving antifungal therapy. METHODS: We initiated a retrospective observational description of patients with candidemia who were treated with antifungal agents. A total of 108 patients diagnosed with candidemia between August 1995 and September 2003 at our teaching hospital were enrolled. Three groups (candidemia with antifungal therapy, candidemia without antifungal therapy, antifungal therapy without candidemia) of patients >18 years of age were evaluated for the presence of thrombocytosis. Platelet administration, pharmacologic or pathologic contributors to thrombocytosis, and other pertinent details related to an elevation of platelet counts were scrutinized. RESULTS: Reactive thrombocytosis was observed in approximately 10% of treated patients with candidemia. Within the subgroup developing reactive thrombocytosis, life-threatening thrombotic complications were uncommon. Mean baseline platelet counts were 393 x 10(3)/mm(3), with a mean peak (695 x 10 (3)/mm(3)) occurring an average of 13 days after initiation of therapy. All patients had resolution within 7 days after therapy. The maximum peak $(1056 \times 10(3)/mm(3))$ was observed in a patient after 14 days of antifungal therapy. The onset of thrombocytosis in this patient was 4 days and lasted 4 days after therapy. CONCLUSIONS: Reactive thrombocytosis occurs during treatment of candidemia. The causative agent (drug vs disease), the risk associated with this reaction, and evaluation of treatment need to be elucidated by a larger epidemiologic study or controlled, prospective clinical trial.

ANSWER 6 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L5reserved on STN

2005561466 EMBASE ACCESSION NUMBER:

Angiogenesis in the human heart: Gene and cell therapy. TITLE:

Tirziu D.; Simons M. AUTHOR:

M. Simons, Departments of Medicine and Pharmacology and CORPORATE SOURCE:

Toxicology, Dartmouth Medical School, Dartmouth-Hitchcock

Medical Center, Lebanon, NH 03756, United States.

michael.simons@dartmouth.edu

SOURCE: Angiogenesis, (2005) Vol. 8, No. 3, pp. 241-251.

Refs: 113

ISSN: 0969-6970 CODEN: AGIOFT

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery

022 Human Genetics 025 Hematology

037 Drug Literature Index 038 Adverse Reactions Titles

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 26 Jan 2006

Last Updated on STN: 26 Jan 2006

The concept of therapeutic angiogenesis - stimulation of new vessels growth to restore blood supply to ischemic tissue has been studied in a number of clinical trials in patients with advanced coronary and peripheral arterial disease. This review discusses the main biological processes underlying new vessel growth and addresses applications of growth factor and cell therapy based on the stimulation of angiogenesis. While still very young and controversial, cell therapy has an enormous potential that is yet to be explored. Multiple questions remain unanswered including the choice of the best cell type, patient selection and the mechanism of action. Nevertheless, much should be expected in this area in the next decade with the likely emergence of new therapies for treatment of ischemic diseases. COPYRGT. Springer 2005.

L5 ANSWER 7 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights

reserved on STN

ACCESSION NUMBER: 2004536240 EMBASE

TITLE: Treatment of head and neck cancer in elderly

patients: State of the art and guidelines.

AUTHOR: Bernardi D.; Barzan L.; Franchin G.; Cinelli R.; Balestreri

L.; Tirelli U.; Vaccher E.

CORPORATE SOURCE: oma@cro.it

SOURCE: Critical Reviews in Oncology/Hematology, (2005) Vol. 53,

No. 1, pp. 71-80. . .

Refs: 48

ISSN: 1040-8428 CODEN: CCRHEC

PUBLISHER IDENT.: S 1040-8428(04)00136-2

COUNTRY: Ireland

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 014 Radiology 016 Cancer

037 Drug Literature Index 038 Adverse Reactions Titles

LANGUAGE: English
SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 6 Jan 2005

Last Updated on STN: 6 Jan 2005

AB Although the majority of head and neck cancers occur between the fifth and sixth decade, their onset in patients older than 60 years is not a rare event. A peculiar characteristic of almost all case series is the lower prevalence of radical treatments among elderly as compared to younger patients, in particular surgery and combined treatment of surgery plus radiation therapy or chemotherapy and radiation therapy. Radiotherapy is a feasible treatment in elderly patients, also in very advanced age groups and, in the era of organ preservation, chemotherapy combined with RT has a paramount importance. Therapeutical planning must be based not only on tumor characteristics, but also on the physiological, rather than the chronological age the patient. The main clinical problem is, therefore, the selection of patients to be

administered anticancer treatment. In patients aged 70 or older, complete geriatric assessment and a multidisciplinary approach are the crucial points. .COPYRGT. 2004 Elsevier Ireland Ltd. All rights reserved.

ANSWER 8 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights L5 reserved on STN

ACCESSION NUMBER: 2005474679 EMBASE

[Stem cell therapy in patients with peripheral arterial TITLE:

occlusive diseasel.

STAMMZELLTHERAPIE BEI PAVK.

Kopp Ch.W.; Steiner-Boker S.; Gschwandtner M.; Minar E. AUTHOR:

Dr. Ch.W. Kopp, Universitatsklinik fur Innere Medizin II, CORPORATE SOURCE:

Abteilung fur Angiologie, Wahringer Gurtel 18-20, A-1090

Wien, Austria. Christoph.kopp@meduniwien.ac.at

Zeitschrift fur Gefassmedizin, (2005) Vol. 2, No. 3, pp. SOURCE:

> 12-15. . Refs: 27

ISSN: 1812-9501

Austria COUNTRY:

Journal; General Review DOCUMENT TYPE: Internal Medicine FILE SEGMENT: 006

> Cardiovascular Diseases and Cardiovascular Surgery 018

Developmental Biology and Teratology 021

029 Clinical Biochemistry

German LANGUAGE:

English; German SUMMARY LANGUAGE:

Entered STN: 8 Dec 2005 ENTRY DATE:

Last Updated on STN: 8 Dec 2005

Autologous bone marrow (BM)-derived stem cell therapy for the induction of AB therapeutic angiogenesis is a potentially limbsaving strategy in patients with chronic limb ischemia and no surgical or interventional option for revascularisation. This review shall present the underlying therapeutic concept and clinical guidelines how to gain profit of the angiogenic potential of BM-derived stem cells. Finally, stem cell mobilization and targeted homing will be discussed as a potential alternative to BM-derived stem cell transplantation.

CAPLUS COPYRIGHT 2007 ACS on STN ANSWER 9 OF 23 L5

2004:565109 CAPLUS ACCESSION NUMBER:

DOCUMENT NUMBER: 141:100449

Methods of treating neurological conditions TITLE:

with hematopoietic growth factors

Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger, INVENTOR(S):

Carola; Sommer, Clemens; Schwab, Stefan; Kollmar, Rainer: Maurer, Martin: Weber, Daniela; Gassler,

Nikolaus

Axaron Bioscience AG, Germany PATENT ASSIGNEE(S):

PCT Int. Appl., 210 pp. SOURCE:

CODEN: PIXXD2

Patent DOCUMENT TYPE:

English LANGUAGE:

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT	KINI	KIND DATE				APPL:	ICAT:	DATE								
WO 2004	A2		2004		Ţ	WO 2	003-		20031231							
WO 2004058287 A8						2004	1021									
WO 2004	0582	87		A3		2004	1216									
W :	ΑE,	AG,	AL,	AM,	AT,	AU,	AZ,	·BA,	BB,	BG,	BR,	BW,	BY,	ΒZ,	CA,	CH,
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	LK,	LR,	LS,	LT,	LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NI,	ЙО,

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NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ,
            TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
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            BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
            ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
            TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
                                         US 2003-659295
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    US 2004141946
                                20040722
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                                            CA 2003-2511294
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                                20051005
    EP 1581249
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             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
                                            BR 2003-17910
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     BR 2003017910
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                                20051129
                                            CN 2003-80110075
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                                20060405
     CN 1756556
                                                                   20031231
                                20060413
                                            JP 2005-509731
     JP 2006512419
                          A1
                                20050630
                                           US 2004-880101
                                                                   20040630
    US 2005142102
                                                                A 20021231
                                            US 2002-331755
PRIORITY APPLN. INFO.:
                                            US 2003-659295
                                                                A 20030911
                                                                W 20031231
                                            WO 2003-IB6446
    The present invention relates to a method of treating neurol.
AB
    conditions in a mammal by administering a hematopoietic growth factor such
    as granulocyte-colony stimulating factor (GCSF) and granulocyte-
    macrophage colony stimulating factor
     (GMCSF). The invention also provides methods of screening for
     compds. that bind to a GCSF or GMCSF receptor found on the
     surface of a neuronal cell; and which provides a neuroprotective,
    neuroproliferative and/or a STAT gene activation activity.
                      CAPLUS COPYRIGHT 2007 ACS on STN
     ANSWER 10 OF 23
                         2004:162774 CAPLUS
                         140:210821
DOCUMENT NUMBER:
                         Cell modulation using a cytoskeletal protein
TITLE:
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L5

ACCESSION NUMBER:

INVENTOR(S):

Losordo, Douglas W.; Kishore, Raj

PATENT ASSIGNEE(S):

Caritas St. Elizabeth's Medical Center of Boston,

Inc., USA

SOURCE:

PCT Int. Appl., 79 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

	PAT	ENT 1	NO.			KIND DATE						ICAT]		DATE				
		- 					-			•			- -					
	WO	20040	01673	39		A2		2004(0226	. 1	WO 2	003 <i>-</i> 0	JS239	78		20	0308	301
	WO	O 2004016739						20050	0414									
		W:	AE,	AG;	AL,	AM,	AT,	AU,	AZ,	BA,	BB,	BG,	BR,	BY,	BZ,	CA,	CH,	CN,
			CO,	CR,	CU,	CZ,	DE,	DK,	DM,	DZ,	EC,	EE,	ES,	FI,	GB,	GD,	GE,	GH,
								IN,										
			LS,	LT,	LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NI,	NO,	NZ,	OM,
			-		•	-		RU,										
								US,										
		RW:	-		-			MZ,								AM,	AZ,	BY,
			KG,	KZ,	MD,	RU,	TJ,	TM,	AT,	BE,	BG,	CH,	CY,	CZ,	DE,	DK,	EE,	ES,
								IE,										
								CM,										
	AU	2003	2579	60		A1		2004	0303		AU 2	003-2	25796	50		20	00308	301
	US	2004	1058	60		Al		2004	0603	,	US 2	003-6	5334(7		20	00308	301
PRIO	RITY	APP	LN.	INFO	. :					1	US 2	002-4	10008	34P	1	2 (00208	301
	WO 2003-US23978 W 20030801																	
AB																		

in a mammal. Practice of the invention generally involves changing activity of the ezrin cytoskeletal protein sufficient to increase or decrease proliferation of the cells. Also disclosed are useful screens for detecting agents capable of modulating ezrin activity. The invention has a variety of useful applications including use in the treatment of diseases associated with unsatisfactory EC proliferation.

L5 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:117103 CAPLUS

DOCUMENT NUMBER: 140:157937

TITLE: Use of erythropoietin

PATENT ASSIGNEE(S): Bahlmann, Ferdinand Hermann, Germany

SOURCE: Ger. Offen., 17 pp.

CODEN: GWXXBX

DOCUMENT TYPE: Patent LANGUAGE: German

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PAT	PATENT NO.						KIND DATE			APPL	ICAT	ION 1	DATE				
DE	1023	4192			Al	_	2004	0212]	DE 2	002-	1023	4192	-	20	0020	726
CA	2493	598			A1		2004	0212	(CA 2	003-2	2493	20030725				
WO	2004	0127	59		A2		2004	0212 [.]	Ţ	WO 2	003-1	EP82	20030725				
WO	2004	0127	59		A3		2004	0603									
WO	2004	0127	59		B1		2004	0708									
	W:	AE,	AG,	AL,	, MA	AT,	AU,	AZ,	BA,	BB,	BG,	BR,	BY,	BZ,	CA,	CH,	CN,
		CO,	CR,	CU,	CZ,	DK,	DM,	DZ,	EC,	EE,	ES,	FI,	GB,	GD,	GE,	GH,	GM,
		HR,	HU,	ID,	IL,	IN,	IS,	JP,	KE,	KG,	KP,	KR,	KZ,	LC,	LK,	LR,	LS,
		LT,	LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NI,	NO,	NZ,	OM,	PG,
		PH,	PL,	PT,	RO,	RU,	SC,	SD,	SE,	SG,	SK,	SL,	SY,	TJ,	TM,	TN,	TR,
		TT,	TZ,	UA,	UG,	US,	UZ,	VC,	VN,	YU,	ZA,	ZM,	ZW				
	RW:	GH,	GM,	KE,	LS,	MW,	MZ,	SD,	SL,	SZ,	TZ,	UG,	ZM,	ZW,	AM,	ΑŻ,	BY,
		KG,	ΚZ,	MD,	RU,	TJ,	TM,	AT,	BE,	BG,	CH,	CY,	CZ,	DE,	DK,	EE,	ES,
		FI,	FR,	GB,	GR,	HU,	IE,	IT,	LU,	MC,	NL,	PT,	RO,	SE,	SI,	SK,	TR,
		BF,	ВJ,	CF,	CG,	CI,	CM,	GA,	GN,	GQ,	GW,	ML,	MR,	NE,	SN,	TD,	TG
AU	2003	2552	90		Al	2004	0223	•	AU 2	003-	2552	20030725					
EP	1526	867			A2		2005	0504	EP 2003-766302						2	0030	725
	R:	AT,	BE,	CH,	DE,	DK,	ES,	FR,	GB,	GR,	IT,	LI,	LU,	NL,	SE,	MC,	PT,
		ΙE,	SI,	LT,	LV,	FI,	RO,	MK,	CY,	AL,	TR,	BG,	CZ,	EE,	HU,	SK	
BR	2003	0129	81.		A		2005	0614		BR 2	003-	1298	1		2	0030	725
CN	1681	526			A		2005	1012	1	CN 2	003-	8221	16		2	0030	725
JP	JP 2006503001						2006	0126	,	JP 2	004 -	5253	22		2	0030	725
NO		A		2005	0418		NO 2	005-	1002		2	0050	224				
US		A1		2005	1208	1	US 2	005-	5224	20050325							
PRIORITY APPLN. INFO.:											002-			i		0020	
							WO 2	003-	EP82	29	1	W 2	0030	725			

AB The present invention concerns the use of erythropoietin for stimulation of physiol. mobilization, proliferation and differentiation of endothelial progenitor cells, for stimulation of angiogenesis, for therapy of diseases connected to a dysfunction of endothelial progenitor cells, and for production of pharmaceutical compns. for treatment of such diseases as well as pharmaceutical compns., which contain erythropoietin and other suitable active substances for stimulation of endothelial progenitor cells.

L5 ANSWER 12 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005021293 EMBASE

TITLE: [Pathophysiology and therapy of reversible posterior

leukoencephalopathy syndrome (RPLS)].

PATHOPHYSIOLOGIE UND THERAPIE DES REVERSIBLEN POSTERIOREN

LEUKOENZEPHALOPATHIESYNDROMS (RPLS).

AUTHOR: Obermann M.; Kastrup O.; Glzewski E.; Maschke M. CORPORATE SOURCE: M. Obermann, Universitatsklinikum Essen, Klin. und

Poliklin. F. Neurologie, Hufelandstrasse 55, 45122 Essen,

Germany. mark.obermann@uni-essen.de

SOURCE: Aktuelle Neurologie, (2004) Vol. 31, No. 10, pp. 481-489.

Refs: 75

ISSN: 0302-4350 CODEN: AKNUAR

COUNTRY:

Germany

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 008 Neurology and Neurosurgery

026 Immunology, Serology and Transplantation

037 Drug Literature Index

LANGUAGE: German

SUMMARY LANGUAGE: English; German

ENTRY DATE: Entered STN: 20 Jan 2005

Last Updated on STN: 20 Jan 2005

Reversible posterior leukoencephalopathy syndrome (RPLS) is a widely AB recognized neurological disorder, considering the increasing number of publications over the past two years. Oedematous cerebral white matter lesions particularly involve the posterior parietal and occipital lobes, but may also affect the brainstem, basal ganglia and cerebellum. This leads to characteristic neurological symptoms such as headache, visual disturbances, nausea and vomiting, altered mental status and seizures. This syndrome is often associated with an abrupt increase. in blood pressure, mainly in patients with eclampsia, renal insufficiency and hypertensive encephalopathy. Immunosuppressive and immunomodulating drugs such as cyclosporine A, tacrolimus, interferone- α and filgastim may also lead to RPLS. A rare variant of RPLS is the isolated brainstem leukoencephalopathy, which is characterized by extensive MRI lesions associated with little clinical symptoms. The respective lesions are best visualized with FLAIR and T(2)-weighted magnetic resonance imaging. They show diffuse hyperintensity generally involving cerebral parieto-occipital regions, but may also selectively affect the brainstem without accompanying supratentorial lesions. Diffusion weighted imaging is an important diagnostic tool to differentiate the mainly vasogenic edema of RPLS from the cytotoxic edema of acute cerebral ischaemia. Appropriate therapy consists of rapid and sustained correction of Symptoms can be completely reversible and MRI lesions may hypertension. show complete remission. Early recognition of RPLS is extremely important, because of its benign prognosis under therapy. Delay of appropriate treatment, however, may lead to permanent damage of affected brain tissue. This review will give an overview of current knowledge of pathophysiology, diagnostic procedures and treatment options on RPLS. Special consideration will be given to reversible isolated brainstem leukoencephalopathy.

L5 ANSWER 13 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2004117498 EMBASE

TITLE: Perspectives for gene therapy in renal diseases.

AUTHOR: Imai E.; Isaka Y.

CORPORATE SOURCE: Dr. E. Imai, Division of Nephrology, Department of Internal

Medicine, Osaka Univ. Grad. Sch. of Med., Suita, Osaka

565-0871, Japan

SOURCE: Internal Medicine, (2004) Vol. 43, No. 2, pp. 85-96.

Refs: 92

ISSN: 0918-2918 CODEN: IEDIEP

COUNTRY: Japan

DOCUMENT TYPE: Journal; General Review FILE SEGMENT: 022 Human Genetics

Urology and Nephrology
Drug Literature Index
Adverse Reactions Titles

039 Pharmacy.

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 25 Mar 2004

Last Updated on STN: 25 Mar 2004

Somatic cell gene therapy has made considerable progress last five years ABand has shown clear success in some clinical trials. In the field of nephrology, both the elucidation of pathophysiology of renal diseases and the development of gene transfer technique have become driving force for new therapy of incurable renal diseases, such as Alport syndrome and polycystic kidney disease. Gene therapy of renal cancer, although its application is limited to advanced cancer, is the front-runner of clinical application. Erythropoietin gene therapy has provided encouraging results for the treatment of anemia in uremic rats and recently progressed to the inducible one in response to hypoxia. Gene therapy for glomerulonephritis and renal fibrosis showed prominent impact on experimental models, although the safety must be confirmed for prolonged treatment. Transplant kidney is an ideal material for gene modification and induction of tolerance in the transplant kidney is an attractive challenge. Emerging techniques are becoming available such as stem cell technology and messenger RNA silencing strategies. We believe that the future of gene therapy research is exciting and promising and it holds an enormous potential for clinical application.

L5 ANSWER 14 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005043742 EMBASE

TITLE: Generalisability in economic evaluation studies in

healthcare: A review and case studies.

AUTHOR: Sculpher M.J.; Pang F.S.; Manca A.; Drummond M.F.; Golder

S.; Urdahl H.; Davies L.M.; Eastwood A.

CORPORATE SOURCE: M.J. Sculpher, Centre for Health Economics, University of

York, York, United Kingdom

SOURCE: Health Technology Assessment, (2004) Vol. 8, No. 49, pp.

iii-117. . Refs: 286

ISSN: 1366-5278 CODEN: HTASFX

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review FILE SEGMENT: 004 Microbiology

018 Cardiovascular Diseases and Cardiovascular Surgery

033 Orthopedic Surgery

036 Health Policy, Economics and Management

037 Drug Literature Index 038 Adverse Reactions Titles

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 10 Feb 2005

Last Updated on STN: 10 Feb 2005

Objectives: To review, and to develop further, the methods used to assess ABand to increase the generalisability of economic evaluation studies. Data sources: Electronic databases. Review methods: Methodological studies relating to economic evaluation in healthcare were searched. This included electronic searches of a range of databases, including PREMEDLINE, MEDLINE, EMBASE and EconLit, and manual searches of key journals. The case studies of a decision analytic model involved highlighting specific features of previously published economic studies related to generalisability and location-related variability. The case-study involving the secondary analysis of cost-effectiveness analyses was based on the secondary analysis of three economic studies using data from randomised trials. Results: The factor most frequently cited as generating variability in economic results between locations was the unit costs associated with particular resources. In the context of studies based on the analysis of patient-level data, regression analysis has been advocated as a means of looking at variability in economic results across locations. These methods have generally accepted that some components of resource use and outcomes are exchangeable across locations. Recent

studies have also explored, in cost-effectiveness analysis, the use of tests of heterogeneity similar to those used in clinical evaluation in The decision analytic model has been the main means by which cost-effectiveness has been adapted from trial to non-trial locations. Most models have focused on changes to the cost side of the analysis, but it is clear that the effectiveness side may also need to be adapted between locations. There have been weaknesses, in some aspects of the reporting in applied cost-effectiveness studies. These may limit decision-makers' ability to judge the relevance of a study to their specific situations. The case study demonstrated the potential value of multilevel modelling (MLM). Where clustering exists by location (e.g. centre or country), MLM can facilitate correct estimates of the uncertainty in cost-effectiveness results, and also a means of estimating location-specific cost-effectiveness. The review of applied economic studies based on decision analytic models showed that few studies were explicit about their target decision-maker(s)/jurisdictions. The studies in the review generally made more effort to ensure that their cost inputs were specific to their target jurisdiction than their effectiveness parameters. Standard sensitivity analysis was the main way of dealing with uncertainty in the models, although few studies looked explicitly at variability between locations. The modelling case study illustrated how effectiveness and cost data can be made location-specific. In particular, on the effectiveness side, the example showed the separation of location-specific baseline events and pooled estimates of relative treatment effect, where the latter are assumed exchangeable across locations. Conclusions: A large number of factors are mentioned in the literature that might be expected to generate variation in the cost-effectiveness of healthcare interventions across locations. Several papers have demonstrated differences in the volume and cost of resource use between locations, but few studies have looked at variability in In applied trial-based cost-effectiveness studies, few studies provide sufficient evidence for decision-makers to establish the relevance or to adjust the results of the study to their location of interest. Very few studies utilised statistical methods formally to assess the variability in results between locations. In applied economic studies based on decision models, most studies either stated their target decision-maker/jurisdiction or provided sufficient information from which this could be inferred. There was a greater tendency to ensure that cost inputs were specific to the target jurisdiction than clinical parameters. Methods to assess generalisability and variability in economic evaluation studies have been discussed extensively in the literature relating to both trial-based and modelling studies. Regression-based methods are likely to offer a systematic approach to quantifying variability in patient-level In particular, MLM has the potential to facilitate estimates of cost-effectiveness, which both reflect the variation in costs and outcomes between locations and also enable the consistency of cost-effectiveness estimates between locations to be assessed directly. Decision analytic models will retain an important role in adapting the results of cost-effectiveness studies between locations. Recommendations for further research include: the development of methods of evidence synthesis which model the exchangeability of data across locations and allow for the additional uncertainty in this process; assessment of alternative approaches to specifying multilevel models to the analysis of cost-effectiveness data alongside multilocation randomised trials; identification of a range of appropriate covariates relating to locations (e.g. hospitals) in multilevel models; and further assessment of the role of econometric methods (e.g. selection models) for cost-effectiveness analysis alongside observational datasets, and to increase the generalisability of randomised trials. .COPYRGT. Queen's Printer and Controller of HMSO 2004. All rights reserved.